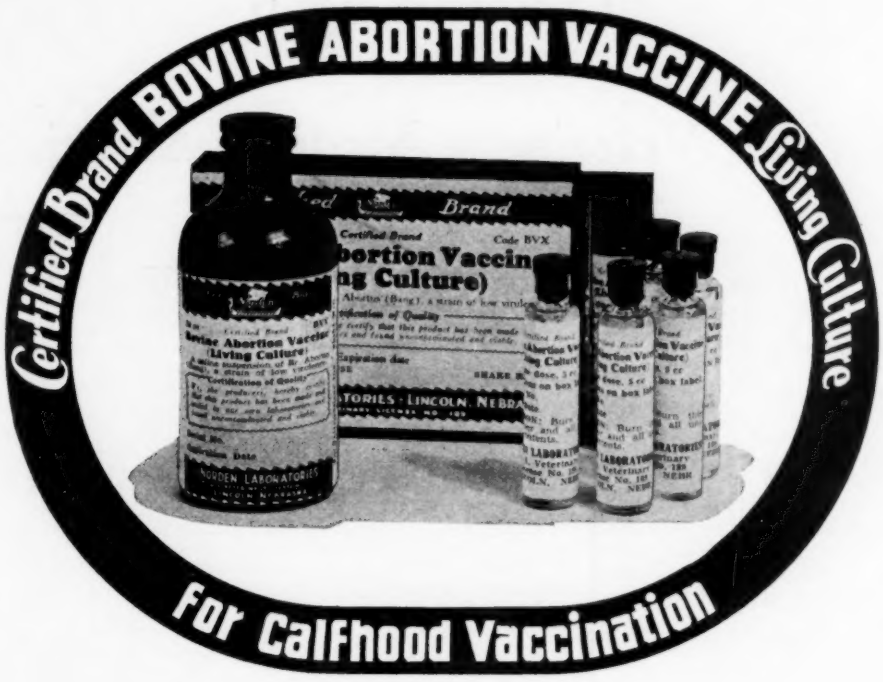
JOURNAL
OF THE
AMERICAN VETERINARY MEDICAL ASSOCIATION

April, 1939

IN TWO PARTS—PART II

Published by
AMERICAN VETERINARY MEDICAL ASSOCIATION
221 N. LA SALLE STREET
CHICAGO, ILL., U.S.A.

Volume XCIV N. S. Volume 47, No. 4



In 5 cc dose recommended by U. S. B. A. I.

Educational Circulars Available

•

Describes nature of Bang's Disease, Modes of Infection, Plans of Control and Procedure.

Calves vaccinated between 4 and 8 months of age are ordinarily negative to agglutination test at breeding age, and immune to Bang's Disease through succeeding pregnancies.

Build Bang's free Herds—Vaccinate the Calves!

Norden Certified Brand BOVINE ABORTION VACCINE is prepared from U.S.B.A.I. Strain No. 19, in accordance with method of Drs. Cotton and Buck. Low-virulent and one-injection treatment. Tested and found to be uncontaminated and viable.

BVX. 6—5 cc . . $1.50 **BVY. 50 cc (10 doses) . . $2.00**

Sales to Graduate Veterinarians Only

NORDEN LABORATORIES

LINCOLN *"The Mark of Quality"* **NEBRASKA**

Official Organ

of the

American Veterinary Medical Association

CONTENTS—

GENERAL ARTICLES:

Induced Cases of Traumatic Gastritis and Pericarditis in Dairy Cattle—R. W. Dougherty 357
Actinobacillosis in Sheep—H. Marsh and H. W. Wilkins 363
Pathology of Calf Pneumonia—W. T. S. Thorp and E. T. Hallman 365
A Method of Preparing Bovine Udders for the Study of Mastitis—O. W. Schalm and C. M. Haring 372
Resistance to Bang's Disease of Cattle That Have Passed Through an Artificially Induced Outbreak and Recovered—B. A. Beach 374
The Use and Value of the Phosphatase Test for Control of Pasteurization—James D. Bohn 376
Quality Milk and Its Control—George H. Hopson 378
Snake Bites Among Domestic Animals—Raymond L. Ditmars 383
Hookworm Disease in Dogs—J. W. Landsberg 389
Skin Diseases Occurring in Both Man and Animals—Howard Fox 398
Some Diseases of the Eyes of Lower Animals: Methods of Examination, Diagnosis and Treatment—Carl F. Schlotthauer 404
Sulfured Soil for Poultry Yards—M. W. Emmel 409
Chemical Changes in the Blood of Swine Infected with Hog Cholera—D. F. Eveleth and L. H. Schwarte 411
Anaphylaxis as Related to Biologic Prophylaxis and Treatment of Animals—John Reichel 418

EDITORIAL 421
APPLICATIONS 428
CLINICAL DATA:

Tibial Fracture in a Horse Treated Successfully—J. F. Thomas 430
Marsh's Disease—A. Savage 431
Fetal Ascites—J. Micuda 432
Trout Poisoning—A. M. McCapes 432
Anthrax in Farm-Raised Mink in Oregon—C. R. Howarth and L. Seghetti 433
Paratyphoid and Trichomonas Infection in Pigeons—W. E. Niemeyer 434
Botulism in Foxes—Norman J. Pyle and Richard M. Brown 436
Fibrosarcoma—R. L. Booth 440
Infectious Equine Encephalomyelitis: Mid-Winter Case—O. L. Osteen 441

LEGAL OPINIONS 443
CURRENT LITERATURE 445
THE NEWS:

Of General Interest, 452; Coming Meetings, 454; Meeting Reports, 455; U. S. Government, 457; Personal Notes, 458.

$4.00 per annum. Foreign $5.00; Canada $4.50 Single Copies 40 cts. in U. S.

Published monthly at 221 N. La Salle St., Chicago, Ill. Entered as Second Class Matter, August 19, 1932, at the Post Office in Chicago, Illinois, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103. Act of February 28, 1925, authorized August 10, 1932. Address all correspondence to American Veterinary Medical Association.

Journal
of the
American Veterinary Medical Association
(Registered U. S. Patent Office)
221 N. La Salle St., Chicago, Ill.

Manuscripts are acknowledged on the same day they are received. Reprints should be ordered in advance. Prices will be quoted upon application.

Please send prompt notice of change of address, giving both old and new. Advise whether the change is temporary or permanent.

Vol. XCIV, N. S. Vol. 47 APRIL, 1939 No. 4

Induced Cases of Traumatic Gastritis and Pericarditis in Dairy Cattle*

By R. W. DOUGHERTY, *Corvallis, Ore.*
Oregon State Agricultural College

PERHAPS many clinicians and practitioners have been impressed with the inadequacy of present diagnostic methods for the determination of early foreign body cases encountered in cattle practice.

According to Udall,[1] "So many of the symptoms of traumatic gastritis are common to those of primary indigestion that an indefinite diagnosis is frequent, especially at the time of the first examination." Since advanced cases are frequently poor surgical risks, an early diagnosis is important to the clinician as well as the patient.

Inadequate or unreliable histories frequently make it difficult to differentiate between acute primary conditions and recurring chronic ones. Certainly, then, some supplemental laboratory tests that practitioners with limited time and laboratory facilities could use would be extremely helpful.

With these facts in mind, a case of traumatic gastritis was induced in a cow with a rumen fistula. This gave a chance to study, at frequent intervals, the hemocytological changes, as well as the pulse rate, temperature, and rate of contractions of the rumen, appetite, attitude, and general condition of the animal. Normal studies of the animal were made for a two-week period, prior to the time that the foreign body was put in place.

PROCEDURE

The first animal used was a Jersey cow, aged seven years, known to be free of foreign bodies for a period of two years, or from the time the rumen fistula was surgically established. During the time, she was used for demonstration purposes for physiology laboratory classes. During the course of these demonstrations, it was necessary to remove part of the ingesta; subsequently, the reticulum was thoroughly explored in search of foreign bodies.

Hematological studies were confined to total and differential leukocyte counts, although a few erythrocyte counts and sedimentation rates were made in the beginning.

Blood samples were taken from the jugular vein by means of a 5 cc. syringe, the barrel of which was moistened with a

*Published as Technical Paper No. 293 with the approval of the Director of the Oregon Experiment Station; contribution of the Department of Veterinary Medicine. Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

saturated solution of sodium citrate. The samples were taken at the same time each day, when the various clinical observations were made. The syringe was rotated constantly from the time the samples were taken until the counts and smears were made. Wright's stain was used in staining the blood smears.

Fifteen normal observations and counts were made over a period of 20 days before the foreign body was inserted. For the beginning, a simple classification of leukocytes was made, using the method proposed by Farley, St. Clair, and Reisinger,[2] differentiating the neutrophils into filament and non-filament types, according to their nuclear structure, that is, if there was any band of nuclear material except the chromatin filament connecting different parts of the nucleus, the neutrophil was classified as the non-filament type.

uneasiness 24 hours after the foreign body was put in place and, 36 hours afterward, she became greatly depressed and refused to eat. She began eating again on June 26 and showed progressive improvement until the wire was removed July 2, at which time her external appearance and behavior were practically normal. During the time of her greatest depression, she showed some of the symptoms usually associated with cases of traumatic gastritis, such as kyphosis, abduction of the elbows, stiffness of gait, with little desire to move about.

Table II compares the average normal figures with those obtained during the first three days following the insertion of the foreign body. After the third day the blood pictures began to approach the normal figure.

Counts were discontinued on July 14, and resumed again on the 17th, the leukocytes

TABLE I—*Normal observations and counts made over a period of 21 days.*

	AVERAGE	MAXIMUM	MINIMUM
Erthrocytes	6,371,066	6,875,066	5,400,000
Leukocytes	5,550	6,950	4,350
Total	23.1%	32%	14%
Neutrophils non-filament type % of total	19.7%	28%	12%
Basophils	0.5%	2%	0%
Monocytes	8.3%	11%	5%
Lymphocytes	61%	66%	53%
Temperature	101.0	101.8	100.2
Pulse	67	70	62

Table I indicates the normal observations and counts made over a period of 21 days, 14 counts being made during this period.

On June 22, the foreign body was forced through the anterior wall of the reticulum so that about an inch of wire protruded through to the peritoneal cavity. The foreign body consisted of a piece of bailing wire, three inches long, sharpened at one end and bent at the other to prevent it from passing on through the wall of the reticulum. The sharpened end was barbed to keep it from working back into the reticulum.

The first blood picture was made 24 hours later and daily readings and observations were continued until July 2, when the wire was removed. The animal showed some

being classified according to the Schilling hemagram.[3] The change was made to this classification so as to permit a better comparison between the kaleidoscopic view of infection in the blood picture and the clinical symptoms.

On July 26, the wire was forced through the wall of the reticulum at 9:00 p. m., and readings and observation were started at 7:00 the next morning. The leukocyte count changed from 7,000 per cu. mm. to 10,400 per cu. mm. There was a marked neutrophilic leukocytosis, the increase being mainly "stabs." There also was a relative decrease in lymphocytes and monocytes.

During the next two days, the animal showed practically no external symptoms,

TABLE II—*A comparison of the average normal figures with those obtained during the first three days after insertion of the foreign body.*

	AVERAGE	24 HOURS AFTER INSERTION OF FOREIGN BODY	2ND DAY	3RD DAY
Erythrocytes	6,371,066	6,763,000	6,483,000	6,000,000
Leukocytes	5,550	10,250	13,250	10,500
Total	23.1%	42%	41%	29.2%
Neutrophils non-filament type % of total	19.7%	79.3%	85%	71.6%
Basophils	0.5%			
Eosinophils	5.5%	5.9%	9.5%	13.3%
Monocytes	8.3%	11.1%	11.0%	7.4%
Lymphocytes	61%	40%	38.5%	48.2%
Temperature	101.0	103.4	102	102.8
Pulse	67	68	65	72

temperature, pulse and contractions of the rumen remaining practically the same. On the assumption that this lack of symptomatic response was caused by the wire being forced through the wall too near the original area of adhesion to the diaphragm, on July 28, at 9:00 p. m., the wire was removed, the adhesions broken down and the wire again forced through the wall of the reticulum.

Table III compares the blood picture made on July 28, before the adhesions were broken down and the wire replaced, and the ones made on the following three days. In making this comparison one must bear in mind that the blood picture on July 28 was made from an animal showing hematopoietic disturbances.

Daily counts showed a gradual return of the blood picture to normal. On August 5, palpation of the wall of the reticulum revealed a firm enlargement, about two inches in diameter, surrounding the foreign body.

Because the cow had shown practically no noticeable symptoms since the time that the wire was first inserted, it was thought that the animal might be developing an immunity against the limited number of organisms and toxins that might invade the peritoneal cavity through these punctures.

On the day that the wire was removed, August 5, a quantity of fluid was obtained from the reticulum and passed through two layers of gauze. Four cc. of this fluid was injected through the wall of the reticulum.

Quite a decided blood response followed, but still the animal showed practically no clinical symptoms. In 15 hours the white count had mounted from 6,600 per cu. mm. to 8,450 per cu. mm., the "stabs" from 7.5 per cent to 56 per cent, and the segmented neutrophils from 1.5 per cent to 8 per cent while the lymphocytes decreased from 79 per cent to 24.5 per cent.

A clinical case suggested that hemocytological changes would be more marked in a case of traumatic pericarditis. Therefore, on September 20, at 10:00 p. m., the wire was forced through the wall of the reticulum and through the diaphragm so that the tip of the wire just touched the heart.

Twenty-four hours later, the animal showed great depression, anorexia, a stiffness of gait, in short, very much the same symptoms that were manifested after making the primary traumatic insult. Two additional symptoms were noted within a few days, a decrease in rapidity and amplitude of the contractions of the rumen and a loss of weight. After the first few days, several sedimentation tests also were made, showing an increase in the sedimentation rate for the first time since the experiment was started.

Since the cow's condition seemed to be getting steadily worse, the wire was removed on October 6, to see whether the animal would recover after showing definite pericarditis symptoms. At this time, extensive adhesions were noted between the wall of the reticulum and the diaphragm.

Wishing to confirm further the original results, a rumen fistula was established surgically, in a grade Jersey cow, aged six years, in good condition and in the early stages of pregnancy.

A few preliminary blood pictures were made before the operation, and several were made afterward. Twenty-four hours after the operation, the total white count had changed from 4,900 per cu. mm. to 8,250 per cu. mm., the stabs from 12.1 per cent to 22.2 per cent, and the lymphocytes from 68.08 per cent to 52.4 per cent.

The animal's appetite was rather poor for the first three days; then her condition steadily improved. Temperature, pulse, and contractions of the rumen remained practically the same. After the third day, the blood picture made a shift to the right. By October 2, the total and differential counts were practically back to pre-operative proportions. When the cow was examined October 5, palpation revealed some adhesions on the wall of the reticulum, but the nail was found lying free in the floor of the reticulum.

TABLE III—*A comparison of the blood picture made on July 28, before the adhesions were broken down and the wire replaced, and those made on the following three days.*

	JULY 28 (9:00 P. M.)	JULY 29 (7:00 A. M.)	JULY 30 (8:00 A. M.)	JULY 31 (8:00 A. M.)
Leukocytes	10,000	13,500	11,250	10,800
Eosinophils	10.0%	10.0%	15.0%	10.8%
Basophils			0.9%	
Myelocytes	5.0%	2.0%	2.6%	2.9%
Juviniles	4.0%	11.0%	2.8%	3.5%
Stabs	18.5%	47.0%	30.8%	25.2%
Segments	6.0%	4.0%	6.5%	6.5%
Lymphocytes	49.0%	21.0%	36.6%	45.3%
Monocytes	7.5%	5.0%	3.2%	5.9%
Temperature	100.8	101.8	101.0	101.2
Pulse	60	68	68	68
Rumenal contractions	3 in 15 seconds	3 in 85 seconds	3 in 80 seconds	3 in 90 seconds

The animal made an uneventful recovery from the operation and the blood-cell elements gradually resumed their pre-operative proportions. Heat loss through the fistula was responsible for a considerable loss in weight. It is necessary to allow the margins of the fistula to heal before applying the rubber plug.

On September 20, palpation revealed a nail through the wall, well circumscribed with adhesions and a small piece of bailing wire lying in the reticulum. These objects were removed before inserting the nail. The first blood picture was made nine hours later and revealed an increase in the total count, from 6,600 to 7,750 per cu. mm., the stabs from 22.3 per cent to 38.5 per cent, the segmented neutrophils from 7.3 per cent to 5.5 per cent, the lymphocytes from 57 per cent to 44 per cent, and the monocytes from 6.5 per cent to 6 per cent.

At this time the nail was reinserted through the wall of the reticulum and frequent blood examinations were made, in an effort to determine just how soon changes might be observed in the blood picture.

Changes were noticeable six hours and 45 minutes after insertion of the wire and were quite marked in 24 hours.

A rumen fistula was established surgically in a grade Jersey, aged ten years, on December 20. The lower commissure of the wound did not heal well and the total white and stab counts remained rather high.

The wire was inserted on February 4, at 7:15 a. m. Six hours later, the total white count showed a decided increase, but little change was noted in the differential count until the 30th-hour sample was taken. The wire was removed on February 14 and, by March 4, the blood counts were quite

similar to those made before the wire was inserted.

On February 9 the counts were resumed on the first cow used in the experiment. A change was made at this time in the method of making smears. Ten cc. of blood was drawn in a clean, dry syringe. Fresh blood smears were made immediately and the remainder of the blood was ejected into a test tube containing 0.1 cc. of a saturated solution of sodium citrate. The blood tube was then placed in a blood shaker for one-half minute and shaken just before the total white count was made.

At this time, the cow was pregnant. The total white and "stab" counts were higher than the initial normal counts. The cow calved on February 22 and the calf died three days later.

On February 28, at 7:25 a. m., a nail was inserted through the wall of the reticulum and counts were made at two-hour intervals. This animal had been free of any experimental procedure for a period of about 4¾ months.

The stab cells showed a marked increase four hours after the nail was inserted. There was a noticeable decrease in contractions of the rumen two hours after insertion of the nail. Outwardly, the only other readily noticeable symptom was a decrease in appetite for about 24 hours.

The nail was removed on March 4, at which time a considerable enlargement could be palpated in the wall of the reticulum in the region of traumatic insult.

This animal became sick on March 19, refusing to eat and showing typical symptoms of traumatic gastritis. A blood picture revealed a neutrophilic leukocytosis, with the young forms predominating. She died on March 25.

Postmortem examination revealed extensive adhesions on the abdominal surface of the diaphragm, a tract containing caseated pus leading from the diaphragm to the pericardial sac, extensive masses of caseated pus and organized adhesions around the pericardial sac, adhesions between the pericardium and epicardium, and extensive adhesions between the parietal and visceral pleurae.

Over a period of eight months, in coöperation with one of the local practitioners, blood examinations were made of about 30 suspected foreign-body cases.

Some of these cows were not operated because of unfavorable blood pictures as well as unfavorable clinical symptoms. Others were not operated because the blood pictures did not indicate infections. All except one of those that were operated recovered. This cow had aborted a few days prior to the operation. Postmortem examination revealed some nephritis. Postmortem changes were too great to permit a thorough autopsy. No foreign bodies were found either at the time the rumenotomy was performed or during the course of the postmortem examination.

In all but one case the blood pictures seemed to corroborate the clinical findings. They seemed to be equally valuable in prognosis as well as diagnosis. Sedimentation rates increased only in samples from the most unfavorable cases.

One smear was sent by mail a distance of 300 miles and was not stained until three days after it was made. The smear stained excellently. Postmortem findings corroborated the blood picture.

DISCUSSION

There are many opinions on the importance of traumatic gastritis and pericarditis in cattle. According to Eber's statistics of 235 correctly reported cases of paresis of the rumen (overfilling of the rumen, primary and secondary gastric atony) in cattle, 17.9 per cent were due to foreign bodies.

Some think that a much higher percentage of the gastric disorders of cattle are traceable to foreign body insults. This, no doubt, will vary in different communities according to the management practices.

It is entirely possible that many cases of so-called atony of the rumen may be caused by foreign bodies in the stomach. Many recurring cases may be due to a shifting of the foreign body in the reticulum,

through the wall and back into the lumen of the viscus, and then through the wall at some other point.

This work has been done in an effort to improve our present methods of diagnosis and prognosis of foreign body cases. It should be used only as an aid to clinical observations, and not as a definite diagnosis.

CONCLUSIONS

1. Marked blood changes were noted in three cows in which foreign bodies were inserted through the wall of the reticulum so that they extended into the peritoneal cavity. The foreign bodies were carried into the reticulum through rumen fistulae.

2. Hemocytological changes occurred in four to six hours after the foreign bodies were inserted.

3. Blood changes included an increase in the total white count, an increase in "stab" cells, the appearance of myclocytes and juveniles in the blood stream, and a relative decrease in lymphocytes.

4. An increase in sedimentation rate was noted in one animal, in which symptoms of pericarditis were induced by forcing the foreign body through the diaphragm until it touched the heart. This animal showed considerable distress.

5. Rumenal contractions decreased as early as two hours after the foreign bodies were inserted.

6. Animals on which the procedure was repeated showed very few visible symptoms. The blood response, however, was quite marked.

7. A comparison between blood pictures and clinical findings on 30 cases indicated that blood examinations are of great value in prognosing as well as diagnosing traumatic gastritis and traumatic pericarditis.

REFERENCES

[1]Udall, D. H.: The Practice of Veterinary Medicine (1933), p. 45.

[2]Farley, D. L., St. Clair, H., and Riesinger, J. A.: The normal filament and non-filament polymorphonuclear neutrophil count: Its practical value as a diagnostic aid. Amer. Jour. Med. Sci., clxxx (1930), pp. 336-344.

[3]Schilling, v.: The Blood Picture and Its Clinical Significance. Translated and edited by R. B. H. Gradwohl. (C. V. Mosby, Saint Louis, 1929) pp. 408.

DISCUSSION

DR. H. C. H. KERNKAMP: I think Dr. Dougherty is to be complimented for the work he has done—correlating blood pictures with clinical manifestations—and it seems to me that in this presentation today, the thing one would have to observe more than a total leukocytic increase, would be a dial. He told us, I think, that the cows vary normally. He had some 5,000, representing a total white count of 5,000, others 7,000, some 8,000, and yet he would imply that this traumatic pericarditis might just give us an increase of 2,000 or 2,500 white blood cells on the total count.

If you were to get that, let us say, without a history correlated with some clinical history, that, of course, is an adjunct; but if we are to go on the count alone, then you would rather have a dial count because the young forms of neutrophils increased tremendously. The change was great in relative proportion.

I would like to give a warning to some of the men. Maybe you are not quite so familiar with the blood studies. Do not become discouraged at first. Yet, when you hear a man giving special attention to a subject like that, he can see things in a blood cell that some of the rest of us do not see until we have given it some careful study. Especially, I think, we have to be careful in differentiating between these young "stabs" and the next stage into the segmented forms, because a lobule or two might sometimes be lying over a definite segmented cell. If we could see the connecting chain between the different lobes and their position—one lying on top of the other—we would call it a "stab," when actually it might be a young segmenting form.

CHAIRMAN DUKES: Have you made experiments on normal cattle?

DR. DOUGHERTY: In this work we were concerned mainly with the changes we could produce in the animals we use; so, of course, we made careful studies of their blood before going into the work. Lately we have started doing some work on the college dairy herd, trying to get some idea of the normal blood count of dairy cows, and it is quite amazing the variations that you get. I think a lot more work of that kind needs to be done before we can talk about the normal. It is rather hard to tell just how many of these cows have never had any trouble. You can readily see why some of these counts may be up and still the cows appear to be normal.

Another thing I should add is this: These slides can be sent for quite a distance and you can still get a good stain. We had one slide sent from the Washington Experiment Station, a distance of 300 miles—it was in the mail three days—and the slide stained just as good as if it had been made a few minutes before. Of course, when you mail these slides, they have to be that way. I tell them to put adhesive tape around each end of the slide, then put the smears in, then tape the slides together. That holds the smears apart and you can send them in a letter. If properly handled, the slides can be sent quite a distance.

Actinobacillosis in Sheep*

By H. MARSH, *Montana Veterinary Research Laboratory, Bozeman, Mont.*
and H. W. WILKINS, *Montana Livestock Sanitary Board, Helena, Mont.*

In recent years, there have been published a number of articles discussing actinobacillosis in cattle and its relation to actinomycosis, but apparently only one reference to the occurrence of this disease in sheep has been made in the literature. Thomas[1] reported the occurrence of actinobacillosis in South African sheep as the result of feeding prickly pear.

In the spring of 1937, a diagnosis of actinobacillosis in sheep was made in this laboratory. A pyogenic infection of the face, occurring in a band of sheep in the Deer Lodge Valley, in Montana, was investigated and specimens were brought to the laboratory. The disease occurred in a band of 2,000 ewes which had been fed hay during the winter. About 60 of these ewes had died as the indirect result of the disease.

The affected sheep were extremely emaciated, due to their inability to consume sufficient feed. The lesions observed were confined to the region of the face. Autopsies on six of the affected sheep revealed no gross lesions in any region except the head. In most cases the lips were enlarged, indurated, partially encrusted with scabs or dried pus, and there was discharge of pus from a number of small openings. There was enlargement of the parotid and submaxillary lymph nodes. In the connective tissue and muscles of the lips and cheeks, and in the lymph nodes, there were multiple abscesses varying in diameter from 5 mm. to 50 mm., containing greenish pus of a tenacious, doughy consistency. Many of the ewes, as observed in the field, showed scab formation on the lips, which was thought to be identical with the lesions of contagious ecthyma, but inoculation tests on lambs failed to demonstrate the presence of soremouth virus. Figures 1 and 2 show the

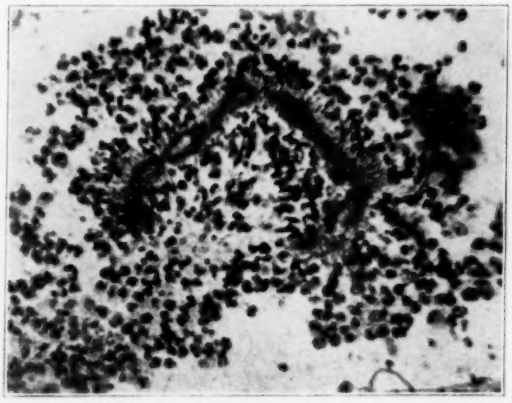
—*After Marsh and Wilkins, 1939.*

Figs. 1 and 2. Two views of a sheep affected with actinobacillosis.

*Contribution from the Montana State College, Agricultural Experiment Station. Paper No. 119. Journal Series. Received for publication, February 25, 1939.

appearance of the faces of two of the affected sheep.

Microscopic examination of the unstained pus from the lip abscesses showed vaguely outlined bodies suggestive of the clubbed ends of the mycelium of Actinomyces as seen in "sulfur granules" of actinomycotic pus, but it was not possible to identify the "rosettes," characteristic of actinomycotic pus. Histological examination of sections of the lips of two of the sheep showed the characteristic lesions of actinobacillosis. There were foci of varying size, consisting of masses of lymphocytes, polymorphonuclear leukocytes, and monocytes. Within these foci there were roughly spherical and crescent-shaped



—*After Tunnicliff*, 1939.

Fig. 3. Section of lesion of actinobacillosis, showing the characteristic radiating club forms (x 450).

"rosettes" similar to those found in actinomycosis, with vaguely outlined, radially arranged clubs on the border. There were no Gram-positive elements demonstrable in these bodies and, therefore, the lesions were considered to be those of actinobacillosis rather than actinomycosis. No giant cells were seen, which is a point of difference between these lesions and the actinomycotic lesions studied in this laboratory. Small lesions of a similar nature were found in sections of a lymph node. Figure 3 shows one of the lesions in a section of an affected lip.

Cultures on serum-agar slants from lip abscesses and from lymph nodes of three of the affected sheep developed Gram-negative rods. This organism and another strain, recovered from a similar case occurring at another place, and studied in detail by E. A. Tunnicliff, of this laboratory, have been identified as *Actinobacillus ligniersi*. The details of the bacteriological work will be published in a separate report.

Although the only published report of actinobacillosis in sheep which we have found is that of Thomas[1] in South Africa, it is our opinion that the same disease of sheep has been described under another name by Christiansen,[2] in Denmark, Magnusson,[3] in Sweden, Jowett,[4] in Scotland, and Thorshaug,[5] in Norway. The causative organism was described by Christiansen in 1917, and given the name *Bacterium purifaciens*. We do not have access to Christiansen's original article, but his work is reviewed by Magnusson. Magnusson, Jowett, and Thorshaug follow Christiansen in attributing similar pyogenic infections to *B. purifaciens*. Magnusson's description of the organism would apply as well to *A. ligniersi*. The symptoms and lesions described by Magnusson correspond to those of the cases observed here, but he does not report any histological findings.

SUMMARY

Actinobacillosis affecting the facial region has been observed in sheep on two premises. In one instance, a large percentage of 2,000 ewes in one band were affected.

It is believed that this disease is probably identical with a disease of sheep described by Christiansen and others in Europe as caused by *Bacterium purifaciens*, which is probably identical with *Actinobacillus ligniersi*.

REFERENCES

[1]Thomas, A. D.: Actinobacillosis and other complications in sheep which may arise from the feeding of prickly pear (*Opuntia* spp.). 17th Rpt. Dir. Vet. Serv. & Anim. Indus., Union So. Africa, 1931, Part 1, pp. 215-229.

[2]Christiansen, M.: Maanedskr. f. Dyrlaeger, xxix (1917), p. 449. Cited by Magnusson.

[3]Magnusson, H.: A contribution to the knowledge of pyaemic forms of infection in sheep. Jour. Comp. Path. & Ther., xlii (1929), pp. 73-91.

[4]Jowett, Walter: Observations on a pyogenic infection of sheep. Jour. Comp. Path. & Ther., xliv (1931), pp. 202-208.

[5]Thorshaug, K.: Byldeske hos sau fremhalt av *B. purifaciens*. Norsk. Vet. Tiddskr., xlvi (1934), pp. 377-387.

Pathology of Calf Pneumonia*

By W. T. S. THORP *and* E. T. HALLMAN, *East Lansing, Mich.*
Animal Pathology Section, Michigan Agricultural Experiment Station

INTRODUCTION

The frequent occurrence of calf pneumonia in a large number of calves presented for autopsy motivated this work. The fact that calf pneumonia is usually a bronchopneumonia seems fairly well established, yet a complete study from a pathological standpoint is definitely lacking.

Since the work of Nocard,[1] in 1901 and 1902, there has been considerable work on calf pneumonia associated with calf scours. In 1917, Hagan[2] described a pneumonia associated with white scours in calves, at which time it was mentioned that in some cases scouring was negligible, while joint lesions and a peculiar type of bronchopneumonia were present. Carpenter and Gilman[3] later stated that the lesions in the lungs varied considerably as to the type of the pneumonia and the duration of the disease. This work, although confined mostly to the etiology of the disease, is significant, as it deals with calf pneumonia as a separate disease. Micrococci, *Corynebacterium pyogenes*, colon bacilli and streptococci were isolated from their cases.

Smith[4] reported a pneumonia in fetuses and calves in which *Brucella abortus* was the causative organism or a predisposing factor when the pneumonia was caused by other organisms. Hallman, Sholl and Delez[5] described the lesions of fetal pneumonia associated with Brucella infections.

In a report made by Clark, covering a period of two years, the economic importance of calf pneumonia to the dairyman was illustrated.[6] In one dairy herd, during the first year, 36 calves out of 50 developed pneumonia, 13 cases resulting fatally. The second year, 17 calves out of 31 developed pneumonia, with eight cases terminating fatally. With a morbidity of approximately 65 per cent and a mortality of approximately 25 per cent for the two-year period, the effect on the dairyman can be easily understood.

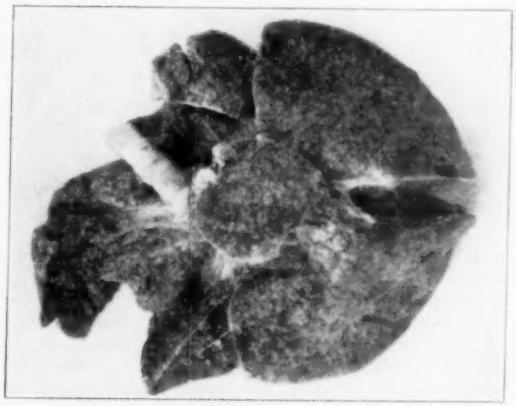


Fig. 1. The ventral aspect of the lungs of a two-month-old calf with bronchopneumonia of three-week duration (1/6 actual size).

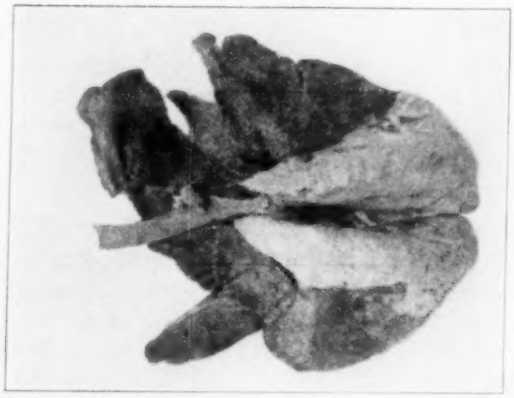


Fig. 2. The lungs from a two-month-old calf with pneumonia of two-week duration. Note the extension of the pneumonia into the diaphragmatic lobes.

METHOD OF STUDY

The materials used in this work were obtained from cases of calf pneumonia brought to the Animal Pathology laboratory for autopsy during the last three years.

*Condensed from a thesis submitted to the faculty of Michigan State College in partial fulfillment of the requirements for the degree of Master of Science in Animal Pathology. Received for publication, April 28, 1938.

Published with the permission of the Director of the Michigan Agricultural Experiment Station as Journal Article No. 323.

Blocks of tissue were taken from various parts of the different lobes, the number depending on the extent of the consolidated areas of pneumonia and their gross variations. All blocks of tissue were fixed in Zenker's solution and embedded in paraffin.

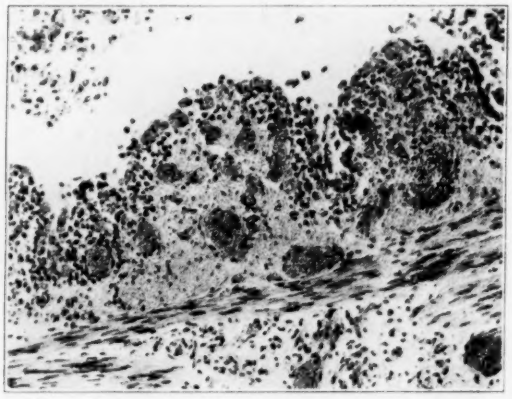


Fig. 3. Section from the mediastinal lobe of the lung illustrated in figure 2, showing an acute bronchitis with desquamation of the epithelium. Note the congested capillaries (x 130).

Blocks from the bronchial and posterior mediastinal lymph nodes also were taken.

Sections were cut 7μ in thickness and stained with hematoxylin and eosin. Mallory's aniline blue stain was used in several cases as a differential stain. Five series of serial sections were made from blocks of previously sectioned material. The lungs were cultured only in those cases which had been dead for a very short time. The bronchial and mediastinal lymph nodes also were cultured.

GROSS EXAMINATION

There are several distinct differences in the gross picture of certain groups of cases. In 21 of the 40 cases studied, there was distinct evidence of a pleurisy, which for the most part was chronic. In two of the cases, there was an acute fibrinous pleurisy. In these cases the pleura covering the cardiac and apical lobes and a small portion of the diaphragmatic lobes showed a fibrinous inflammation. The pleura in these cases varied in thickness from 2 mm. to 1 cm. In eight of the 40 cases studied, there were numerous small abscesses in the lung which ranged in size from 3 mm. to 1 cm. in diameter. In five cases, the bronchial type of pneumonia was manifested by the gross picture of the disease (fig. 1).

There was considerable variation in the extent of consolidation noted in the diaphragmatic lobes of either lung. In gross the pneumonia in the left lung was consistently one of longer duration than that of the right lung. Numerous areas were grayish yellow in color and appeared quite atelectatic as compared with the pneumonic lobes of the right lung of the same animal. The pneumonia in the left diaphragmatic lobe in many cases did not involve so much of the lobe as the usually more acute, consolidated portion of the right lobe. However, in one case the entire left lung was consolidated and only a small portion of the right diaphragmatic lobe was not pneumonic. The posterior mediastinal and bronchial lymph nodes were very much enlarged in 23 of the 30 cases. When numerous petechial hemorrhages were present in the

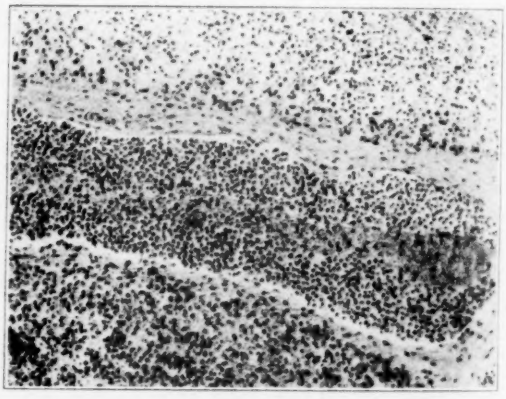


Fig. 4. Section from the lung of a four-month-old calf with a pneumonia of 18-day duration, showing distention of one of the lymphatics of an interlobular septum with fibrin and leukocytes (x 130).

lymph nodes, they were more numerous usually in the mediastinal nodes than in the bronchial lymph nodes, which were enlarged and yellowish white. All the cases studied showed a bilateral pneumonia.

TYPICAL CASES

The following protocols illustrate two typical cases of pneumonia:

Case 4: A calf, which was one of a set of triplets and the first to become sick.

Just prior to the pneumonia, the calves were changed to another nurse cow and developed a mild scours. This calf became sick on March 14, 1935, and died on April 5, 1935, at the age of one month. Upon the first signs of pneumonia (temp., 104.8), the animal was given 40 cc. of calf-scours antiserum. Three days of improvement followed.

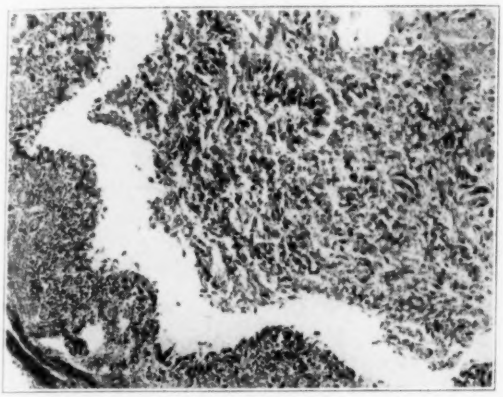


Fig. 5. Section from the lung of a six-month-old calf with a pneumonia of three-day duration, showing an acute bronchitis with desquamation of the epithelium. Note the large number of epithelial cells in the lumen of the bronchus (x 105).

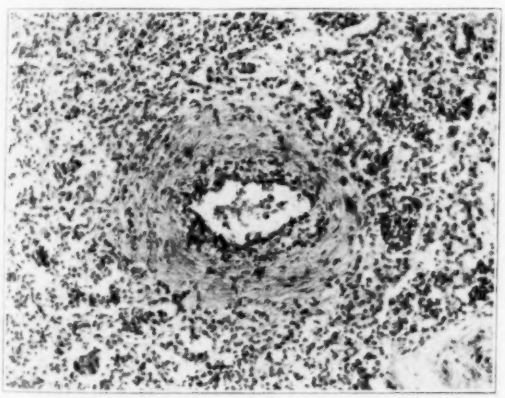


Fig. 6. Section of the lung of a 14-week-old calf with a clinical history of pneumonia of one-week duration, showing considerable productive tissue changes with fibrosis in the wall of a respiratory bronchiole (x 130).

On March 25, 1935, the calf was noticeably worse (temp. 102). Loss of weight and persistent coughing were accompanied by accelerated respiration and diarrhea. Daily injections of calf-scours antiserum were made until April 5, 1935.

Gross examination: The cardiac and apical lobes of both lungs show a grayish-red consolidation with a pleura studded by numerous reddish-brown areas ranging from 1 to 2 mm. in diameter, which suggested necrosis. The diaphragmatic lobes were consolidated only in the anterior one-third, which appeared much the same as the adjacent cardiac lobes. The mediastinal lobe was similar in appearance to the cardiac lobes. Throughout the cardiac and apical lobes of both lungs, many small abscesses, 5 mm. to 1 cm. in diameter, appeared just beneath the pleural surface. The abscesses seemed more numerous toward the hilus of the lobe. The involved portions of the left lung showed more gray areas indicative of a more abundant cellular exudate.

Upon section of any of the lobes, there exuded from the smaller bronchi and bronchioles a foamy purulent exudate which was considerably thicker than that from the large bronchi. Many abscesses similar to those noted under the pleural surface were seen upon section. If a lobe of the lung showing numerous abscesses on the pleural surface had been cut so that the main bronchi and some of their branches were divided longitudinally, several of the abscesses appeared at the bifurcations of the bronchi and at the terminations of the small bronchi. On the cut surface of the anterior lobes, many opaque, yellowish patches were observed which appeared to include an entire lobule. The mediastinal and bronchial lymph nodes were enlarged and had a distinct zone of hemorrhage around the peripheral border.

Microscopic examination: The pleura was slightly thickened. The interlobular septa were wide and edematous and the lymphatics were distended with mononuclear phagocytes. In many of the small bronchi the mucosa was markedly congested and accompanied by considerable desquamation of the epithelium. Productive tissue changes were very much in evidence around the small bronchioles. These were accompanied by considerable mononuclear infiltration. No productive tissue changes were noted in the walls of the alveoli surround-

ing the bronchioles. There were many necrotic areas 0.5 to 1 cm. in diameter. The alveolar walls were very badly congested and the alveoli were filled with an exudate consisting of mononuclear phagocytes and a small number of polymorphs in a network of fibrin. In many instances the extensive mononuclear exudate was predominant. The lymph nodes contained a large number of polymorphs in their sinuses.

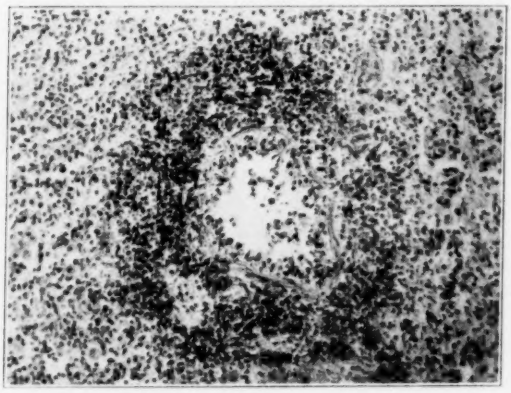


Fig. 7. Section from the lung of an eight-week-old calf with a pneumonia of twelve-day duration, showing a small bronchiole with marked cellular infiltration of the peribronchial tissue (x 150).

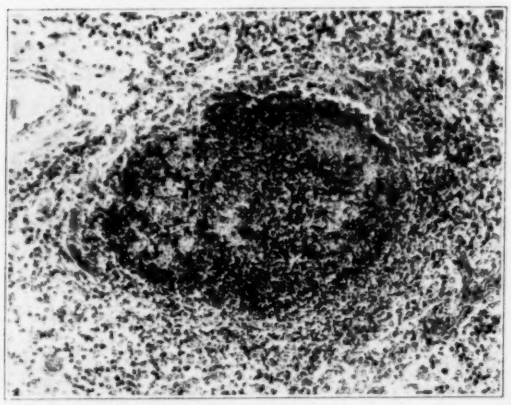


Fig. 8. Section from the lung of a ten-week-old calf with a pneumonia of eleven-day duration, showing suppuration of the wall of the bronchiole and necrosis of the cells of the exudate in the lumen (x 150).

Case 23: A female Holstein, two months old, which had pneumonia for two weeks prior to death.

Gross examination: There was a pneumonia of the diaphragmatic, cardiac, and apical lobes of both lungs (fig. 2). The anterior portion of the right cardiac and all of the right apical lobes appeared very atelectatic. About one-half of the right diaphragmatic lobe was in a state of reddish consolidation. The anterior one-third of the left diaphragmatic lobe was in a state of grayish-yellow consolidation. All of the pneumonic areas of the left lung appeared very atelectatic. Upon section of the more acute areas in the left diaphragmatic lobe, a mottled, grayish consolidation appeared scattered over the cut surface. The mediastinal lobe was entirely consolidated and very similar to the lobes of the left lung. The interlobular septa appeared thickened and edematous. The mediastinal and bronchial lymph nodes were enlarged and edematous.

Microscopic examination: The pleura was thickened. The interlobular septa were edematous and their lymphatics were very much distended. The apical and cardiac lobes of both lungs showed a severe bronchitis with much congestion and desquamation of the epithelium (fig. 3). In the apical lobes there was evidence of considerable productive tissue changes in the walls of the respiratory and terminal bronchioles. The alveoli in the cardiac and apical lobes of both lungs were filled with a cellular exudate consisting mostly of mononuclear phagocytes and a small number of polymorphonuclear leukocytes. The areas of pneumonia in the anterior portion of the right diaphragmatic lobe did not show so many productive tissue changes as the apical and cardiac lobes. In this lobe there was considerable congestion of the alveolar wall accompanied by an abundant serous exudate in the alveolar sacs. In a small number of alveoli, there was a fibrinous exudate surrounded by cuboidal-like cells. Many of the alveoli contained large numbers of giant cells. There was some increase in the number of polymorphs in the lymph sinuses.

Bacteriological examination: A streptococcus of the Beta type was isolated from this case.

DISCUSSION

There was a considerable variation in the microscopic pathology in a number of the

cases studied. Twenty-five cases of the 40 showed productive tissue changes of the pleura, in 15 of which there was also edema of the interlobular septa, which contained many distended lymphatics (fig. 4). The thickening of the pleura was due to a fibrinous pleurisy in some instances and in others it was due to productive tissue changes. Thirty-five of the cases showed

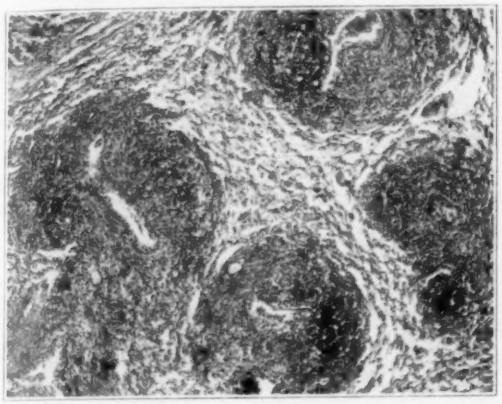


Fig. 9. Section from the lung of a four-month-old calf with a clinical history of pneumonia of two-week duration, showing a subacute bronchopneumonia with considerable atelectasis (x 85).

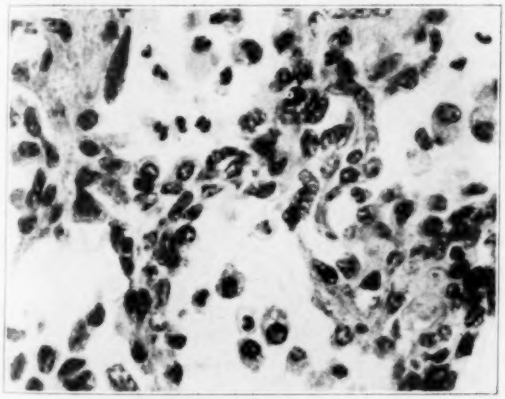


Fig. 10. Section from the lung of a four-month-old calf with a pneumonia of eighteen-day duration, showing an increase in the number of fibroblasts in the alveolar wall (x 550).

an acute bronchitis which ranged in severity from a moderate mononuclear infiltration of the mucosa to a marked congestion with much desquamation of the epithelium (fig. 5). Only one case showed any evidence of a chronic bronchitis.

In most instances, the acute bronchitis was accompanied by much desquamation of the epithelium and congestion of the mucosa. In 18 cases, there was evidence of a chronic bronchiolitis, which was manifested by marked productive tissue changes in the stroma of the mucosa and in many instances the peribronchial tissue (fig. 6). Sixteen of these cases were associated with an acute bronchitis. This would seem to indicate that the infectious agent probably affected the respiratory bronchioles first. The lesions in the respiratory and terminal bronchioles were very similar to those mentioned by Boyd[7] and MacCallum[8] in their descriptions of bronchopneumonia, especially streptococcal pneumonia. In streptococcal pneumonia there is usually more injury to the small bronchioles and surrounding alveoli than to the bronchi.

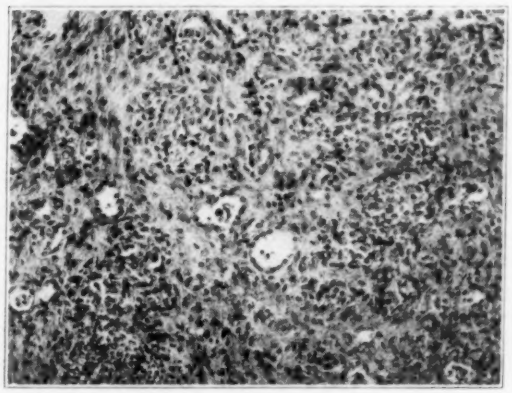


Fig. 11. Section from the lung of a four-month-old calf with a pneumonia of eighteen-day duration, showing marked productive tissue changes in the alveolar wall (x 140).

A cellular reaction around the bronchioles (fig. 7), was noted in some cases, while others showed a necrosis of the bronchioles (fig. 8). The pathology involving the alveoli varied considerably throughout the 40 cases studied. In eight cases of subacute bronchopneumonia, there was a very marked atelectasis (fig. 9), although in most cases some evidence of atelectasis was usually present. Nine cases of the 40 showed productive tissue changes to a variable extent in the alveolar walls (fig. 10). In those pneumonias ranging in duration from two to three weeks, there was considerable evidence of

productive tissue changes in the cardiac and apical lobes. This condition was more pronounced in the left apical and cardiac lobes. This also was true for productive tissue changes in the small bronchioles.

The majority of the alveolar walls were usually very much congested, yet a few contained large numbers of polymorphonuclear leukocytes and some mononuclear cells. In those cases in which the pneumonia was of

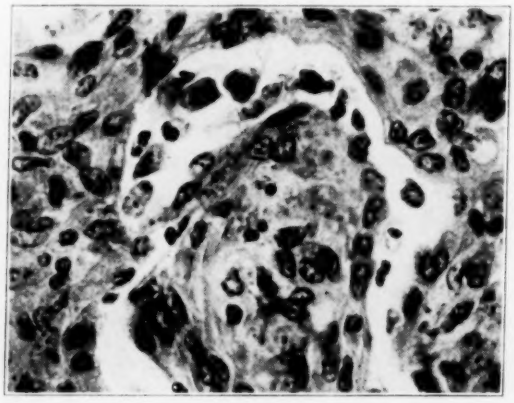


Fig. 12. Section from the same case illustrated in figure 11, showing an organized exudate in the alveolus (x 650).

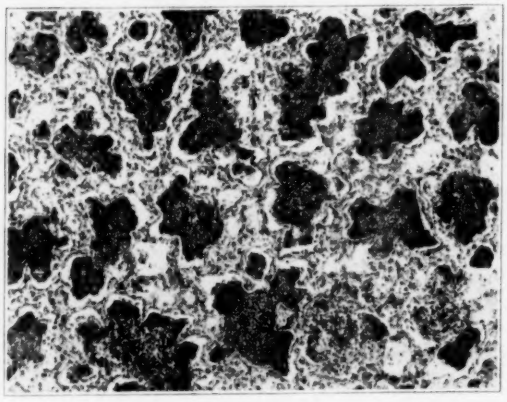


Fig. 13. Section from the lung of a seven-month-old calf with a pneumonia of three-day duration, showing an area in which practically all of the alveoli contain a fibrinous exudate (x 150).

long duration, some alveoli were lined with cuboidal cells and showed evidence of productive tissue change in the alveolar walls. The predominating alveolar exudate was cellular, although serous and fibrinous exudate may be present in variable amounts. In five acute cases the exudate was predominantly serous. In three cases there was fibrosis of the alveolar wall (fig. 11) and organization of the alveolar exudate (fig. 12). In ten cases of the 30, there was much necrosis and abscessation.

In a number of the very acute cases, there was such an outpouring of serous and fibrinous exudates that the pneumonia resembled lobar in type (fig. 13). When a large number of sections were made from these cases and a closer study made, the condition was diagnosed as acute bronchopneumonia. The presence of alveolar pores was indicated by the fibrinous exudate which passed through the alveolar wall into

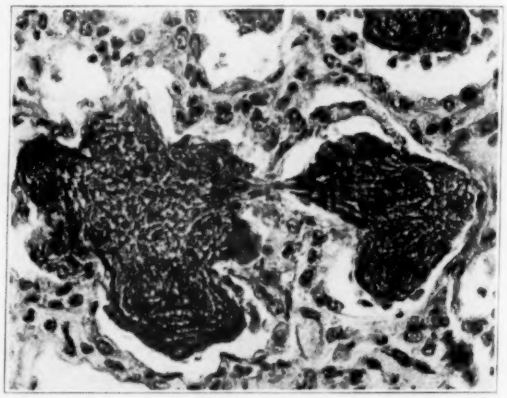


Fig. 14. Section from the case illustrated in figure 13, showing the continuity of the exudate through an alveolar pore (x 650).

the adjacent alveolus (fig. 14). Therefore, it was easily understood how a serous or fibrinous exudate which contained large numbers of organisms could spread the infectious agent from one alveolus to another. In eight cases the pneumonia was associted with an acute bronchiolitis such as that illustrated in figure 15, and in another group of five cases with bronchitis there was an infiltration of a large number of mononuclear phagocytes into the stroma of the mucosa (fig. 16).

The bacteriological findings in this investigation were similar to those of Carpenter and Gilman. The organisms isolated most consistently were: a streptococcus of the Beta type, *Escherichia communior*, and

a staphylococcus, usually *Staphylococcus aureus*. Streptococci were isolated in 13 cases. In four of these the streptococcus was the only organism isolated. In five cases it was associated with the Escherichia organism and in four cases with a staphylococcus. In eleven of the 24 cases cultured, *E. communior* was isolated, in five of which it was associated with streptococci and in five with staphylococci. In four of the cases the staphylococcus was the only organism isolated. A streptococcus and a staphylococcus were present in the majority of cases in which many necrotic and abscessed areas occurred.

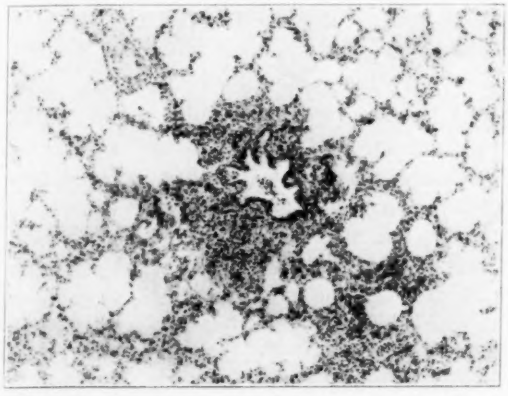


Fig. 15. Section from the lung of a one-month-old calf, showing a terminal bronchiole with a recent bronchopneumonia (x 100).

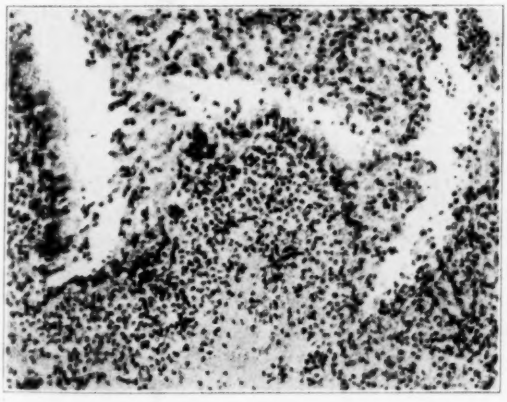


Fig. 16. Section from the lung of a three-month-old calf with a pneumonia of eleven-day duration, showing a recent bronchitis with considerable infiltration of mononuclear phagocytes into the stroma of the mucosa (x 150).

SUMMARY

1. Forty cases of calf pneumonia were studied in gross and microscopically for pathological changes. Twenty-six of these cases were also studied bacteriologically.

2. Three variations were noted in the pathology of these cases: (1) an acute pneumonia, which progresses very rapidly, with the animal dying within several days, without evidence of any productive tissue changes but usually with a serofibrinous exudate filling the alveoli; (2) an acute pneumonia superimposed on a chronic bronchiolitis or chronic bronchopneumonia in which a large part of both lungs is consolidated, accompanied by a marked fibrinous pleurisy, and (3) the cases in which large numbers of abscesses and necrotic areas are present in the lung.

3. That the pneumonia was consistently bilateral appears well established.

4. It was shown by the gross and microscopic studies that the pneumonia usually begins in the apical lobes and that the pneumonia is consistently of longer duration in the left lung than in the right.

5. Evidence in a large number of cases suggests that accompanying injury to the respiratory bronchioles is prior to a bronchitis of the small and large bronchi.

6. Streptoccoci of the Beta type and *Escherichia communior* were considered as probable causative agents in the disease.

REFERENCES

[1]Nocard, E.: A new pasteurellose: White scours and lung disease of calves in Ireland. Amer. Vet. Rev., xxv (1901-02).

[2]Hagan, W. A.: The etiology and mode of infection in white scours of calves. Corn. Vet., vii (1917), p. 263.

[3]Carpenter, C. M., and Gilman, H. L.: Studies in pneumonia in calves. Corn. Vet., xi (1921), p. 111.

[4]Smith, T.: Pneumonia associated with *Bacillus abortus* (Bang) in fetuses of new-born calves. Jour. Exp. Med., xli (1935).

[5]Hallman, E. T., Sholl, L. B., and Delez, A. L.: Observations on the pathology of *Bacterium abortus* infections. Mich Agr. Exp. Sta., Tech. Bul. No. 93.

[6]Clark, C. F.: Calf pneumonia. Rpt. Div. Vet. Sci., Mich. State Coll. (1936).

[7]Boyd, W. A.: Text Book of Pathology. (2nd ed., 1934.)

[8]MacCallum, W. G.: The pathology of the pneumonia in the United States Army camps during the winner of 1917-18. Mono. Rockefeller Inst. for Med. Res., No. 10 (April 16, 1919).

A Method of Preparing Bovine Udders for the Study of the Pathology of Mastitis*

By O. W. SCHALM *and* C. M. HARING, *Berkeley, Calif.*
Veterinary Science Division, University of California

Although much knowledge is available concerning the clinical, chemical and bacteriological aspects of mastitis, its pathology has been accorded relatively little attention. This neglect probably arises in part through insufficient knowledge concerning the histology of the normal mammary gland and also from the fact that the unfixed bovine udder is rather unsatisfactory for gross pathological study. It is so unwieldy and flabby that it is difficult to obtain slices of sufficient thinness and evenness to permit a thorough examination.

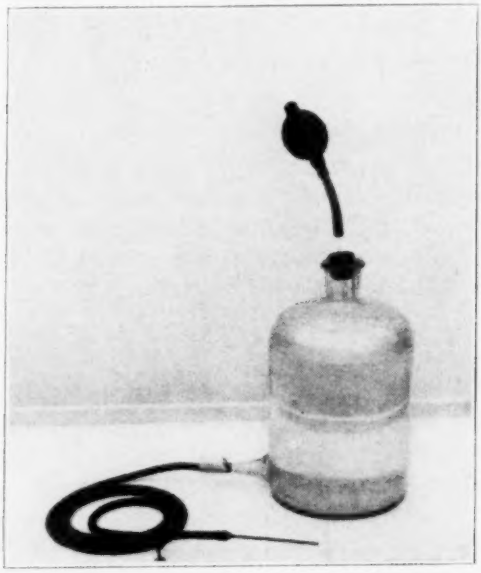


Fig. 1. Apparatus for injecting formalin into the duct and vascular systems of the udder.

In order to obtain a fuller knowledge of mastitis, a study of the gross and microscopic histology of both normal and diseased mammary glands is necessary. To this end, the authors have developed a technic of handling the bovine udder which makes feasible such a study and permits a thorough examination of all parts of the mammary gland.

This technic may be described as follows: The entire udder is removed at slaughter, care being taken not to cut into the gland tissue. After milking out as much of the secretion as possible, the duct and vascular systems are injected with 20 per cent formalin. The apparatus used for the injection is shown in figure 1. It consists of a 5-liter aspirator bottle, containing 20 per cent formalin and fitted with a rubber stopper pierced by a glass tube, to which is attached a rubber bulb with valves for compressing air upon the formalin. A rubber tube, with a blunt needle at one end, is attached to the outlet at the bottom of the bottle.

To fix an udder, the vascular system is injected first. This is accomplished through the pubic artery of each mammary half. As soon as the formalin is observed to escape from the stumps of the large vessels, they are ligated. The injection is continued until all the large vessels have been so closed. The duct system of each quarter is then injected through the tip of the teat until the quarter is distended and tense. The injected udder is stored in 10 per cent formalin until thoroughly hardened. This requires from two to six weeks, depending on its size.

After the udder has hardened sufficiently to insure retention of its shape during subsequent handling, the halves are separated along the median line and then trisected by cutting through the middle of each quarter from the tip of the teat to the base of the gland. The pieces are tagged as follows: RF, RC and RR, or LF, LC and LR, indicating whether right of left front, center

*Presented at the annual meeting of the Conference of Official Research Workers in Animal Diseases of North America, Chicago, Ill., November 29, 1938.

or rear portions of the half. The next step consists of reducing each piece to serial slices of from 3 to 5 mm. in thickness in an electrically driven meat-slicer (figure 2). Tags, numbered consecutively and bearing identification of the mammary half, are attached to the slices of each piece, which are then wrapped together in cheesecloth and stored in formalin until it is convenient to study them.

Each quarter of the udder is a separate and distinct gland and should be studied as such. It is necessary, therefore, to segregate the slices of the center piece of each

Fig. 2. Electrically driven meat-slicer for reducing the udder to a series of slices.

mammary half into those belonging to the front or rear quarter. Since the dividing line between the two quarters is indistinct, the segregation of the slices can be only approximate, unless a stain, such as hematoxylin, has been added to the formalin used to inject the duct system of one of the quarters. This will give the parenchyma a purplish-brown hue in contrast to the light gray or brown of the unstained quarter.

It is not necessary to study an udder immediately after it has been sliced. It may be done whenever convenient, for the udder can be preserved permanently as long as it is kept submerged in formalin. Histological sections prepared from udders preserved as long as three years have shown no deterioration of the finer histological structures.

SUMMARY

A method is described whereby the entire bovine udder may be preserved permanently, in formalin, without material change. By means of an electrically driven meat-slicer, it can be reduced to serial slices of from 3 to 5 mm. in thickness, making possible a minute examination of all portions of the gland. The method is particularly suitable for use in the collection and long-time preservation of normal and pathological mammary glands for teaching purposes.

Beef Gains Increased by All-Summer Grazing

The cheapest beef-cattle gains usually come from pasture grasses. More of these inexpensive gains may be made by supplementing grasses that ordinarily furnish grazing only in the spring and fall with a pasture plant that may be grazed in the summer, studied by the U. S. Department of Agriculture in coöperation with the Missouri Experiment Station show.

At the Sni-a-Bar farms near Kansas City, Mo., rotation grazing of bluegrass pasture, supplemented in midsummer with Korean lespedeza pasture, produced an average of 107.9 pounds of gain per acre. Unsupplemented bluegrass pasture, grazed in rotation, produced only 71.8 pounds and when grazed continuously, only 64.2 pounds.

Furthermore, reports the U. S. Bureau of Animal Industry, cattle grazing on bluegrass supplemented in midsummer by lespedeza pasture made an average gain per head of 70.6 per cent more than cattle on the other two pastures.

A woman visitor to the London Zoo asked a keeper whether the hippopotamus was a male or a female.

"Madam," replied the keeper sternly, "that is a question that should be of interest only to another hippopotamus."—*Reader's Digest.*

Resistance to Bang's Disease of Cattle That Have Passed Through an Artificially Induced Outbreak and Recovered*

By B. A. BEACH, *Madison, Wis.*
Wisconsin Agricultural Experiment Station

The literature on Bang's disease is voluminous. There seem, however, to have been relatively few observations on cows that have passed through outbreaks of the disease and either escaped infection or recovered therefrom. Rettger and White[1] conclude that "infection as indicated by serological tests is in the adult relatively constant." They report on seven animals that reacted to the agglutination or complement-fixation test, or to both, that ceased after a time to react. In a later paper, White, Rettger and McAlpine[2] report on five animals that gave positive reactions, later becoming negative. Fitch, Boyd and Delez[3] report on one animal that became positive to both agglutination and complement-fixation tests, later became negative, and then returned to positive. The animal was slaughtered because of sterility, hence her final status as regards serological reactions was not determined.

All of the observations that have come to our attention have been on herds in which the infection had existed for some time. In such herds, there is seldom any change from positive to negative. Those positive animals that go negative usually do so during the first year after the initial infection. Rettger and White[1] believe that positive-reacting cows are apt to become negative at or near the time of calving or aborting. After three or four weeks, such animals react positively again. We have made observations on two herds. Infection was by the conjunctival method. Two drops (approximately .06 cc.) were deposited in the conjunctival sac by means of a dropper. The inoculum was a pooled 48-hour growth of three Brucella cultures standardized to ten times tube 1 McFarland's nephelometer. These cultures were highly virulent, as shown not only by guinea pig inoculation but also by the fact that a large part (75 per cent) of the susceptible animals in both herds actually aborted.

HERD I

Fourteen cows were under observation. They had been part of a herd of 44 animals in which *Brucella abortus* infection had been artificially induced.[4] Two of the 14 cows evidently escaped infection, for they neither reacted nor aborted. The twelve others reacted in varying degrees, but all were considered positive. Four of them reacted in no higher than the 1:50 dilution, one in the 1:100, four in the 1:200, and three in the 1:400 dilution. Eight weeks was the longest any of the animals was as high as 1:400. The titre of these animals returned to negative, or nearly so.

Thirteen of this number were actually infected. One cow, which happened to be one of the two noted above that actually escaped initial infection, was too far advanced in pregnancy and, consequently, was withdrawn from the herd. The other escape promptly became infected after exposure and aborted. Why these two cows, one of which subsequently was proved susceptible, escaped initial infection is an interesting subject for speculation. The remaining twelve calved normally. This was in spite of the fact that 75 per cent of the controls aborted.

At the time of calving, colostrum milk from each quarter and uterine fluid were taken. The milk was centrifuged and the cream and sediment from each quarter injected intraperitoneally into guinea pigs.

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

The uterine fluid likewise was injected into these test animals. Eight weeks later, the guinea pigs were slaughtered and their blood tested for the presence of agglutinins. Any showing titre were cultured.

With one exception, Brucella was not demonstrated, showing that twelve of the 13 animals were probably not eliminating the organisms. The exception was cow 36. The left rear quarter was found to harbor Brucella. This infection was evidently transitory, as shown by the fact that, during two subsequent pregnancies, the animal was negative. This cow was slaughtered on March 27, 1934, at which time the udder, supramammary lymph-gland, and uterus were searched carefully, both by guinea-pig inoculation and cultural methods, with negative results.

HERD II

In herd I there were seven cows* that returned to normal titre. This made the nucleus for another herd for experimental purposes. Twenty-four heifers were added from herds negative to tests for Bang's disease. One of the seven was eliminated because of an intercurrent infection. This herd was handled in the same manner as herd I. Following infection, the titres of these six cows rose slightly, from 1:25 to a maximum of 1:100. Four of the six calved normally. The two others were not pregnant.

CONCLUSIONS

1. Approximately 28 per cent of two herds artificially infected with Bang's disease recovered, as shown by the fact that the blood titres returned to normal.

2. Eighteen of 19 such cows proved resistant to an artificial Brucella infection that caused abortions in 36 of 47 controls (76 per cent).

3. One cow that had passed through a natural outbreak of Bang's disease and recovered is included in the 18 cows mentioned above. She behaved in exactly the same manner as the others, as far as could be determined.

*One of this number was a cow that had passed through a natural outbreak of Bang's disease.

REFERENCES

[1]Rettger, L. F., and White, G. C.: Conn. Agr. Exp. Sta. Bul. 93 (1918).

[2]White, G. C., Rettger, L. F., and McAlpine, J. G.: Conn. Agr. Exp. Sta. Bul. 123 (1924).

[3]Fitch, C. P., Boyd, W. L., and Delez, A. L.: Report of experimental work in the control of bovine infectious abortion. Jour. A. V. M. A., lxxxv (1929), n. s. 28 (2), pp. 215-229.

[4]Hart, E. B., Hadley, F. B., and Humphrey, G. C.: Wis. Agr. Exp. Sta. Res. Bul. 112 (1932).

Prices of Farm Products

The Bureau of Agricultural Economics looks for moderate improvement in the domestic demand for farm products this spring and summer. A slight change for the better in the outlook for foreign demand is also noted. Farm products prices in domestic markets declined about 4 per cent during the past winter, but recently have been holding relatively steady.

Ballot Shows Most People Favor Lean Cuts of Beef

Given a chance to choose between fat, medium and lean cuts of beef, International Livestock Exposition visitors viewing the United States Department of Agriculture exhibit showed a decided preference for the lean.

Of the more than 1,600 persons voting, 54 per cent favored the lean cut, 31 per cent chose the medium cut, and the remaining 15 per cent liked the fat cut, according to O. G. Hankins, in charge of meat investigations for the Bureau of Animal Industry.

Approximately 70 per cent of the persons voting for the lean cut gave as their reason the economy in higher proportions of lean meat. Nearly 50 per cent of those voting for the medium cut did so because they considered it to have about the right proportions of fat and lean. Palatability factors influenced 35 per cent of those voting for the fat cut, while another 18 per cent preferred this cut because they thought it had the most "quality."

The Chicago exhibit was the first of Department of Agriculture exhibits in which vistiors are being given the opportunity to express their preference on meats.

The Use and Value of the Phosphatase Test for Control of Pasteurization*

By JAMES D. BOHN, *Sheffield Farms Co., New York, N. Y.*

For years, the dairy industry and public health officials have felt the need for some dependable test to check the efficiency of pasteurization of any given sample of milk. As a rule, the only proof of pasteurization was the chart of the recording thermometer on the pasteurizer, and the accuracy of the indication was in turn largely dependent upon the integrity of the operator. Furthermore, charts do not indicate mechanical inaccuracies or the addition of raw or underpasteurized milk, or contamination with underheated foam.

Some plants today are capable of pasteurizing in excess of 50,000 pounds of milk per hour and many have a capacity of 10,000 pounds per hour. Even with the newer types of pasteurizing apparatus and a fair degree of sanitary inspection and control, and although there are comparatively few chances of "slips" in the pasteurizing process, the need for such a test remains. The ideal test would necessarily be dependent upon a normal constituent of milk, present in abundance, the heat resistance of which is at the threshold of the temperature of pasteurization. The enzyme phosphatase is such a substance.

The enzyme phosphatase is always present in normal raw milk in large quantities. It has been reported that the phosphomonoesterase content of cow's milk varies during the lactation period. However, the test is usually applied to market milk and variation due to individual cows or the stage of lactation would be eliminated. This enzyme is almost completely devitalized by the heat treatment required for the pasteurization of milk. It seems almost incredible that such a constituent could be found in milk.

The phosphatase test was developed in 1935, by Kay and Graham, in England. In the United States, the original procedure has been modified by Gilcreas and Davis, of the New York State Department of Health, and Scharer, of the New York City Department of Health. The test is based upon the action of the enzyme phosphatase on a buffered substrate, containing disodium phenyl phosphate. Upon incubation, the enzyme, if present, will hydrolyze the phenyl phosphate radical of the buffer substrate solution, producing free phenol. The presence of phenol is detected by the addition of a very sensitive phenol reagent. The intensity of color is proportional to the amount of enzyme present.

The Gilcreas-Davis modification is considered to be a quantitatve procedure. The greatest limitation to this method is the time, materials and skill necessary to its performance. Therefore, this method probably will be used by the dairy industry only in sections of the country where local health officials use it as a check.

The Scharer modification appears to have the greatest possibility, as it is relatively simple, expedient and inexpensive. Two methods for conducting this test have been developed: an accurate laboratory method using phenol color standards and requiring one hour for completion, and a field test for non-technically trained personnel, which requires about 15 minutes for completion.

The laboratory test roughly consists of the addition of 1 cc. of milk to 10 cc. of buffer substrate and incubating for one hour at 98° F. After incubation, samples are boiled for five minutes and then cooled in ice water. Add 0.1 cc. of basic acetate solution and shake immediately. Allow to stand for one or two minutes. Filter. To 5 cc. of clear filtrate add 0.25 cc. borate buffer. Add two drops B.Q.C. Shake gently. The blue color develops within 15 minutes. The color is then compared with simple phenol color standards and reported as units, a unit being the amount of enzyme which, un-

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

der conditions of the test, would produce the color equivalent of 1 gamma of phenol. This procedure will detect the addition of 0.1 to .01 per cent raw milk.

The field test consists roughly of the addition of 0.5 cc. of milk to 5 cc. of buffered substrate and incubating for ten minutes at 100° F. After the incubation period, remove and add six drops B.Q.C. Shake well. Allow to stand for five minutes. The appearance of any blue color denotes improper pasteurization. The sensitivity of this test can be increased greatly by extracting the blue color, if present, with neutralized normal butyl alcohol. This procedure makes it possible to detect the addition of 0.5 per cent of raw milk in properly pasteurized milk or cream, in less than 20 minutes. The same sample tested by the Kay-Graham method would require 18 to 24 hours incubation. Thus, one can see the advantage of the Scharer modifications.

The field test makes it possible for the laboratory man in the plant to check the pasteurization of all products before bottling or, at least, before delivery. The average milk dealer interested in safeguarding dairy products from the quality and human health standpoint cannot afford to place underpasteurized products on the market when such a valuable test is available and can be easily applied in routine control.

The field test also has a psychological and educational value. The plant employe or plant operator, having seen that the test can detect the presence of improperly pasteurized milk, will be more careful to see that the products are fully pasteurized. It is more convincing to see the results of a test run at the plant than merely to receive a verbal or written notice that pasteurization operations are not correctly performed.

The Scharer test can be applied to dairy products other than milk and cream with slight modifications. The test can be used to check cheese, butter, cottage cheese, buttermilk, chocolate drink and ice cream mix.

When first checking the products of any given plant for pasteurization by the phosphatase test, many startling defects, unknown to the operator, may come to light. Some of the more common defects are dead ends, leaky inlet and outlet valves, underheated foam contamination, inaccurate recording and indicating thermometers, recording clocks running fast, inaccurate temperature controls and standardizing with underpasteurized milk or cream.

In conclusion it may be said that the phosphatase test for determining the efficiency of pasteurization is one of the most important tests developed since the inception of pasteurization for safeguarding dairy products from the quality and human health standpoint. The phosphatase test is not only welcomed by those in the sanitary control of milk but by the industry as well. After all, the important consideration for the industry man is the factor of safety in the use of pasteurized products. Thus, it behooves the industry to do everything possible so that, when a bottle of milk goes out under the label "pasteurized milk," they can be certain it is completely and adequately pasteurized. Although the test probably is not perfect in its present form, it will be modified from time to time as experience dictates and as needs for new applications arise. At the present time, however, it offers the most accurate and sensitive means for determining the efficiency of pasteurization. Therefore, if milk control officials and the dairy industry coöperate in the use of the test and attempt through its use to remedy any existing defects either in equipment or methods, it can be but a benefit to both.

The first three stomachs of the ruminant —the recticulum, the rumen and the omasum—are but a complicated termination of the esophagus, in other words, of the mechanism of deglutition.

Ruminants are the least aggressive of the higher animals, because they are descendants of the timid fauna of the prehistoric ages, which had to snatch a meal in the open and then hide away from their predatory contemporaries to complete the process of mastication.

Quality Milk and Its Control*

By GEORGE H. HOPSON, *Brooklyn, N. Y.*
Kings County Medical Milk Commission

During the past 30 years, milk control has been concerned primarily with the elimination of all diseases which are carried or transmitted through milk. In other words, the sole interest of health authorities was the safety with which the milk could be used by the consuming public. Many of our leading agricultural and daily papers have been prone to publish reports of the numerous outbreaks of septic sore throat, scarlet fever, typhoid fever, diphtheria and other diseases which have been milk-borne, the result being that the American public has been made extremely milk safety-conscious.

The above conditions have necessarily brought about almost universal pasteurization of milk in cities of over 10,000 population, the greatest asset the milk industry has ever had. The phosphatase test, which is now being used as a routine measure to determine whether or not milk has been properly pasteurized, is one more safeguard for the milk-consuming public. We may honestly say that by making milk safe we have achieved the greatest factor for the United States, the largest milk-producing and consuming country in the world. The *National Business Magazine* states that 4,800,000 dairy farms, owning 25,000,000 dairy cattle, represent one-fifth of the farm income. Certainly no one can dispute that safety has brought about the increase in this growing industry.

During the early period of milk control, quality was not overemphasized. Milk, as it was delivered to the receiving station, was tested for butterfat content and adulteration as often as the state inspector could make his rounds, which was generally once a year. Milk-houses were seldom found, and cooling was with the old aerator or not at all. Stable cleanliness was simply shoveling the manure out of the stable window or down in the cellar, to be drawn out in the spring. Stable floors were mostly of wooden planks. Open ceilings and few windows were generally found. Cattle were seldom clipped or groomed. Washing of the cow's udder was unheard of. Utensils were seldom washed and sterilized in an efficient manner. Today dairy conditions, technic and methods have greatly changed for the better from those of yesterday. Even now the dairy industry has plenty of room for improvement of conditions under which milk should be produced.

The public is becoming more and more milk quality-conscious. Hardly a day passes that we do not receive a letter from a physician or a mother asking about the quality of a certain milk. The most common questions asked are, "What is the difference between grade A and grade B milks?" or, "Is vitamin-D milk grade A or grade B?" Many times questions are asked relative to the difference between several distributors' milks. Doctors are always interested in the health of the animals, inquiring whether or not the herds are tuberculin and blood tested, examined for mastitis, and how often. Methods and equipment on the farm are also of special interest to them.

The members of the Kings County Medical Society are loyal supporters of safe, clean, quality milk. Recently one of the journals published by the Society quoted the following definition of quality milk by Dr. M. J. Rosenau, of Harvard University:

> Quality milk is clean milk of uniformly low bacteria content, free from dirt and pathogenic organisms. It is produced from healthy cows, free from tuberculosis, Bang's disease, mastitis and all other maladies, and it is handled by healthy employés, using sanitary methods and clean and sterile equipment and utensils. Finally, such a safe milk must also be relatively high in nutritive value, with an ample content of butterfat and total solids and an

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

abundance of normal vitamins, the content of some of which in milk is definitely influenced by the ration fed the cow.

To me, this is the manner in which a quality milk should be produced and it places the problem of doing so squarely in the lap of the veterinary profession.

The first item of major importance is the health of the cow. A milk supply is no better than the health of the animals from which it is produced. We who are interested in the production of certified milk request that the cattle be tuberculin-tested semi-annually and that all additions be tested at the time of purchase unless originating from a fully accredited herd. Today, bovine tuberculosis is nearly eliminated as a public health menace. The veterinary profession and the United States Department of Agriculture must be given credit for this accomplishment even though it was sponsored as an economic control.

Brucella Abortus Infection

At present, many states are making decided efforts to control *Brucella abortus* infection in cattle. This disease is both a public health and an economic problem. This is a second opportunity for our profession to demonstrate its importance in controlling a disease which is communicable to man.

In raw milk supplies, *Br. abortus* infection in cows is a most important control problem. Herds should be blood-tested at least every 120 days and all suspicious animals should be tested every 30 days. Many producers, who are anxious to establish and maintain clean herds, test their entire dairies every 30 days. All replacements are tested before purchase and then 30 days after entering the herd. Calfhood vaccination may be the answer to our abortion problem. Here in the East, Dr. L. J. Tompkins, of the Sheffield Farms Company, has been doing some very fine work along this line.

Mastitis

This disease is also one of public health and economic importance. It has caused the dairy farmer the greatest financial loss of any of the diseases encountered. This disease can be reduced to a minimum, as has been shown by Dr. D. H. Udall, of Cornell University, by classifying the herds, placing the infected animals by themselves, and milking them last.

On our certified farms especial attention is given to udder health. In the opinion of our Milk Commission, it is impossible to produce a good quality milk from mastitis-infected animals, even though the causative organism most commonly encountered is not pathogenic for man.

All milking cows are examined once a month for mastitis by our farm veterinarians. We encourage the use of many of the several biological tests in conjunction with the examination. The milk from each quarter of every cow is examined before each milking by the milk foreman. An animal showing any sign of abnormal milk is immediately removed to the hospital barn and must be examined by a veterinarian before she is allowed to return to the milking line. Nearly all of the farms are now taking individual and group cow samples which are plated on blood-agar to determine the presence of hemolytic streptococci. Other farms are taking individual milk samples from all cows which show any deviation from the normal and these are examined by the plate and Breed methods for leukocyte count and the presence of streptococci. A standard plate count is also made of these samples. (A healthy udder is most important, for it is here that the milk is secreted.)

One of the large distributors of grade A pasteurized milk in New York City, realizing the importance of keeping abnormal milk from its supply, has adopted a program of more frequent physical examinations. A marked difference has been noted in the bacterial content as well as improvement in the flavor of the milk.

In 1929, Dr. F. D. Holford presented a paper before the International Association of Dairy and Milk Inspectors. He stated:

> The most important part of the dairy cow from a public health standpoint is the udder. It is very important that this organ should be in a healthy condition, for it is the fountain head of nature's most wonderful food upon which many infants

and children are wholly dependant for their sustenance.

Since the bovine udder plays so important a part in quality milk, is it not reasonable to say that the supervision of such should be solely under the veterinary profession?

During the year 1937, we tested over 2,670 consumers' doorstep samples of certified milk in our laboratory. Of this number, only eight counts were above 10,000 colonies per cc. In every case but one, the condition was brought about by high-count cows. Abnormal milk of this type is difficult to detect and many times experienced fore-milkers are unable to discover it. On the average market-milk dairy, mastitis-infected cows are responsible for over 5 per cent of the high counts. We find the thybromol test very effective in tracing high-count cows in our dairies. It has proven extremely valuable in goat herds where mastitis is seldom found but high-count goats are not uncommon. The organism which most generally has proven to be the cause of high counts in goat's milk is a staphylococcus.

Other maladies for which we should look carefully are foot-and-mouth disease, variola, anthrax and actinomycosis.

Next in importance is the care of the animals:

1. Milking animals should be provided with stalls of sufficient length, width and partitions to keep them from injuring one another's udders by stepping on teats. Ample bedding is also very important, as it not only makes the animal more comfortable but also aids greatly in promoting cleanliness and in protecting the udders from cold floors. Rubber mats are being used on some farms and are proving valuable in preventing large hocks and sore feet. These mats are also a considerable saving on bedding.

2. All milking cows should have their posterior quarters well clipped during the stabling period. On certified farms we encourage all-over clipping of the animals. This facilitates the grooming and aids greatly in keeping sediment out of the milk.

3. Washing of the udder and posterior parts of the animal is most essential to the production of quality milk. If an individual, sterile towel is not available for each animal, along with ample, warm chlorine water, all efforts should be given to dry brushing of the udder. The usual type of damp cloth found on the market milk farms is a piece of burlap bag or some other discarded cloth saturated with filth. Bacteriological counts made of samples of these cloths run up into uncountable millions. Efficient washing and drying of the udder is extremely important in the control of sediment in milk. After the animals have been washed, they should remain standing until milked, thus preventing them from becoming soiled.

HEALTHY EMPLOYÉS

It is essential that all employés be in good health if we are to have and maintain healthy animals. Past history has shown that the initial cause for septic sore throat and scarlet fever outbreaks has been due to employés who were sick and yet continued to milk and care for the animals, thus infecting one or more of the cows. All producers should be constantly reminded of the importance of having none but healthy employés in the dairy room as well as in the stable. Periodical medical examinations of all milk handlers, together with laboratory examinations of body specimens, has proven successful and practical on certified farms. On market milk farms this would not prove to be practical. Education, therefore, must be the main line of approach.

METHODS AND EQUIPMENT

Methods and equipment are the next important steps. Safe milk from healthy cows can soon become contaminated if the equipment and methods are poor. It is unnecessary to have expensive equipment but it should be of good, sound construction, easy to take apart, clean and sterilize. Milking machines are most difficult to keep clean and may be the cause of much trouble if not properly cared for. Surface coolers are

an efficient means of cooling but are very difficult to keep clean and are not practical on the average farm. Milk pails and strainers should be of the seamless, solderless type, as they are much easier to keep clean. The less surface over which the milk must pass, the better are the chances for low bacterial content and good flavor.

The milking operation should be conducted as quickly and quietly as possible. The milk should then be strained and rapidly cooled to a temperature of at least 50°, at which it should be held until delivered to the consumer. We have very little trouble with sediment, as we place all our emphasis on the cleanliness of the animal and the stable, rather than on the efficiency of the strainer. Our Commission desires clean milk, not cleaned milk. When sediment does appear, it is sometimes very difficult to find the cause. The following have been the most common causes in our experience:

1. Cows not clean.
2. Cows not properly clipped.
3. Udders not properly washed.
4. Dirty bedding.
5. Rust or sediment in water used for steam sterilization.
6. Improper construction of equipment.
7. Improper storage of clean equipment.

Sterilization of Equipment

Proper cleaning and sterilization of equipment is sadly lacking on the average dairy farm. This alone is a cause of great financial loss to the dairyman for he is unable to attain bacterial premiums. Efficient sterilization may be achieved by the following methods:

Dry heat at 200° F., for 30 minutes, for all metal equipment which can be dismantled and placed in a sterilizer, such as milking pails, teat-cup shells, strainers, cans, small surface coolers.

Steam heat at 200° F., for 30 minutes, is recommended for bottles and holding tank.

Hot water at 190° F. is also very efficient for sterilization of metal holding-tanks, sanitary pipe lines, etc.

Chlorine sterilization is also very widely used but does not seem to be so efficient as dry heat, steam or hot water.

The nutritive value of milk must depend to some extent upon the type and quality of the food given the cow. Today, with the vast knowledge available on harvesting and storing of grains in their succulent stages, we must necessarily add to the nutritive value of the milk from animals fed this food. Many of the farms under our supervision store all their roughage in the green state, so that throughout the year the cattle will have rations which are well balanced and rich in vitamins, carotene and other food substances.

I have touched very lightly on a few points which I believe are essential for the production of quality milk, but I have failed to say anything about the producer. His attitude is most important, for, if he has not the desire to produce a high grade of milk, all the score cards, rules and regulations and veterinary inspections in the country will be of no avail. This condition is prevalent in many grade A sections. Why? If a majority of the farms delivering their milk to a certain factory want to make grade A milk, it is mandatory for the minority to secure the necessary equipment or otherwise leave their market. This group is usually the poorest grade of dairymen in any grade A section. The quality of a milk can always be measured by the interest of those who are responsible for its production.

Supervision and Control

A well equipped laboratory is also important and a necessity in milk control. It should have facilities for making total plate counts, blood-agar plates, tests for *Escherichia coli* in milk and water, and many other tests used in milk laboratories.

It is all very well to have certain methods and standards for the production of a quality milk, but it is also necessary to have the proper persons to supervise and help the producers to carry out these measures.

In other words, the inspector should be one who has a knowledge of veterinary medicine, bacteriology, chemistry and some engineering ability. The veterinarian interested in this type of work is the most suitable. Such men as Hollingsworth, Holford, Hall, Corbin and Hardenbergh certainly have shown their interest in the milk industry as well as the importance of the veterinarian in milk control.

The medical profession, federal, state and local health authorities all acknowledge the fact that the veterinarian should be the logical person to supervise the field of milk sanitation. It is up to each and every one of us who is interested in milk control to give superior service if the public and those in charge of public health activities are to maintain their confidence in us.

DISCUSSION

DR. JACOB P. MAUNEY: I would like Dr. Hopson to tell us how he goes about making group tests, that is, his technic with cows having disorders.

DR. HOPSON: Well, most of our tests are individual cow samples, but if there are any group samples taken, they are usually taken by the drip method, or something of that sort; that is, through an open valve and a pipe line, or something, from a certain number of cows. Most of the work that we do in our laboratory is on individual cow samples, and they are usually taken by washing the udder and disinfecting the teat, and so forth.

The big cattle ranches of the North American plains and the pampas of Argentina have been called the marvels of large-scale animal production. Yet, Amon, Egyptian of the twelfth century, B. C., owned 420,000 cattle and had 86,000 cowboys to watch over them.

What sheep do with their spare time has been discovered by the Sonora (Texas) Ranch Experiment Station. "Detectives" sent out by the Ranch observed that the average sheep day is 13.2 hours. Thirteen per cent of the time the sheep spend in walking around. One-third of 1 per cent is devoted to licking salt and the same time to drinking water. The sheep's big job, however, is eating. Half of the waking time is consumed in that occupation.

Soil and Air Temperatures Limit Growth of Grasses

A three-year study by the U. S. Department of Agriculture, in coöperation with the Missouri Experiment Station, helps to explain why bluegrass is a "northern" grass, why the growth is retarded in the summer and why Bermuda grass grows best in the South. The grass specialists expect the results of this rather technical study—conducted under controlled soil and air temperatures ranging from 40° to 100° F.—to help them solve some of the problems of grazing rotations, pasture mixtures, and all-season grazing. The temperature variations were used to approximate the wide temperature variations in the United States.

Although Kentucky bluegrass made a good above-ground growth at air temperatures of 40° and continued growing even at 90°, the roots grew best at soil temperatures of 60° and stopped growing at 80°. This explains summer "dormancy" of bluegrass.

Canada bluegrass reacted much as did Kentucky bluegrass, except that the best temperatures for both root and herbage growth were about 10° lower than for the Kentucky bluegrass.

Bermuda grass did not begin "normal" growth until soil and air temperatures were about 60°, but both roots and herbage grew well at 100° temperatures, the highest used in the study. On the other hand, Bermuda grass was severely injured by 40° temperatures.

Orchard grass made its best above-ground growth at air temperatures of 70°, grew slowly at 80° and stopped at 100°. Orchard-grass roots grew best when the air and soil were 60° to 70°, but continued growth at 80° in the lower levels—eight to 16 inches. This explains why orchard grass will grow farther south than bluegrass. Although bluegrass makes good herbage growth at a higher temperature than orchard grass, its root growth is stopped by even moderately high soil temperatures.

Snake Bites Among Domestic Animals*

By RAYMOND L. DITMARS, *New York, N. Y., Curator of Mammals and Reptiles, The New York Zoölogical Park*

The treatment of snake bite has been considerably clarified and remedial measures have been greatly improved in recent years. As accidents are rather frequent among various domestic animals and range stock, it is of interest to review conditions as indicated in memoranda gathered by the writer.

First, it is well to note the character of a snake bite and, particularly, the types of poisonous snakes inhabiting North America. These are rattlesnakes, the copperhead snake, water moccasins and coral snakes—grossly speaking, four types.

Fig. 1. The fangs of a rattlesnake. These teeth are hollow, with orifice at the tip, like hypodermic needles. They fold back against the roof of the mouth when the jaws are closed.

Rattlesnakes are found in all of the states except northern Maine, the copperhead in the greater part of the region east of the Mississippi, with extension to Oklahoma and Texas, and the water moccasin primarily in the southeastern states. These snakes have a pair of long, canaliculated teeth, which are open at the tips, somewhat resembling hypodermic needles. Situated in the anterior part of the upper jaw, these teeth are attached to movable bones and fold against the roof of the mouth when the jaws are closed. They connect with the poison-containing glands in the temporal area.

In the snakes mentioned above, the poison is powerfully hemotoxic and causes great swelling. The coral snake of the southeastern states is much smaller than the others. A species of this snake is found also in the southwestern states. They are neurotoxic in the effect of their bites but rarely do they attack domestic animals.

When a poisonous snake bites, it drives its fangs into the victim and the venom is therewith injected. If the serpent is large, the poison is injected relatively deep. A five-foot rattlesnake has fangs approximately one-half inch in length.

The venom is not circulated through the body as quickly as most people believe. A rapid swelling following the bite tightens the tissue area and constricts the channels of absorption. With the swelling comes a rush of serous fluid to the affected area, diluting and to a certain extent neutralizing the toxin in its extension. The larger the body bulk or blood content of the victim, the less the danger, although the amount of venom administered by a big snake may overwhelm the largest victim, unless remedial measures are prompt.

Two Remedial Measures

Two such measures are now available. While the life of the animal may be saved with one or the other, it is far better to employ both. Since a snake bite involves the injection of poison, the affected area must be drained. A small cupping outfit, which is inexpensive but highly efficacious,

*Received for publication, October 7, 1938.

is used in this operation. Small incisions are made into the fang wounds and the poisoned blood is drawn out. Even several hours after an animal has been bitten, it may not be too late to drain a considerable amount of poison, which, as stated, circulates rather slowly.

The attendant remedial measure is the injection of neutralizing antivenin. This is a sterile horse serum of less concentration, and less cost, than that produced for human use. It is a relatively new treatment.

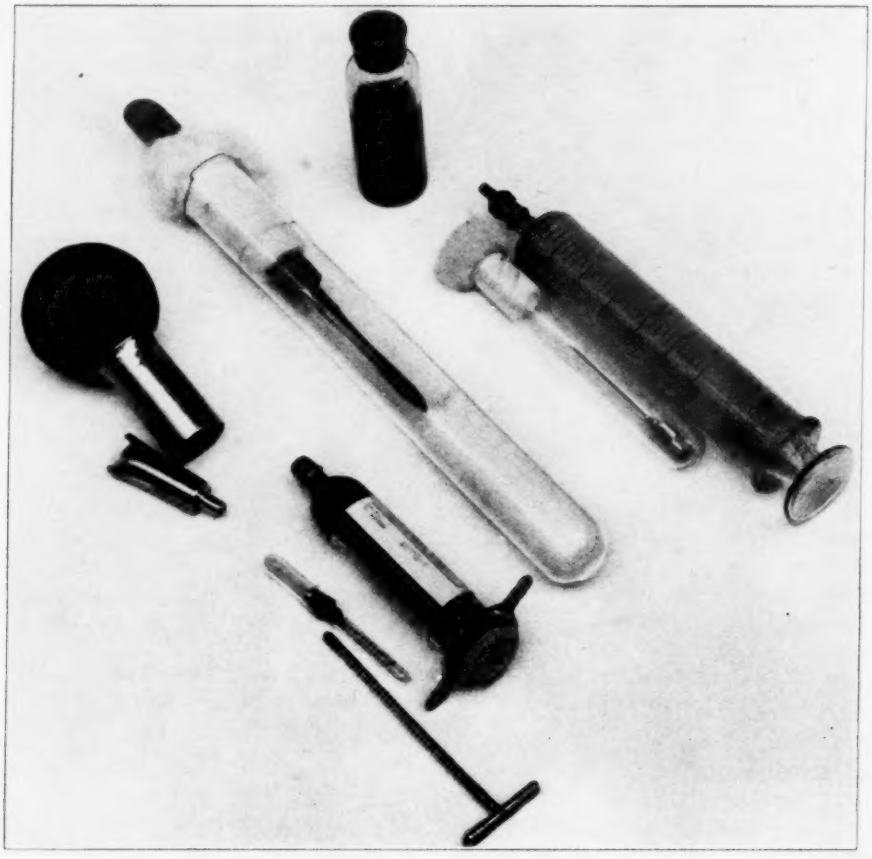


Fig. 2. Implements for treating snake bite. From left to right: suction bulb with two types of applicators and, beneath, loaded Antivenin syringe with needle and handle; center, sterilized scalpel; top, bottle of serum and type of veterinary syringe used where large doses are indicated.

There is an important consideration involved in the administration of antivenin in dogs. Accidents to valuable hunting dogs, particularly in the southeastern states, are disturbingly frequent. On this subject, the writer is indebted to Dr. Thomas S. Githens, of Glenolden, Pa. Quoting Dr. Githens:

> In order to make antivenin available for use in domestic animals at a price lower than that of the syringe package for human use, we put up unconcentrated antivenin in 50 cc. bottles. Of this, 1 cc. neutralizes 1 mg. of standard venom, so that the total immunizing power is greater than that of the 10 cc. syringe, each cc. of which neutralizes 3 mg. This unconcentrated serum is available only for the Nearctic Crotalidic Antivenin, used in North America. For animals bitten by Bothropic snakes and Crotalus terrificus (Central and South American), only the human package is available.
>
> As to dosage, we have not much information, but it is certain that dogs, probably on account of their small size, require relatively larger doses than adult humans, while cattle and horses seem to be more

readily protected. We have only 18 reports of cows and horses treated by antivenin, and all recovered. On the other hand, reports received here of 164 native pit vipers (rattlesnakes, copperhead and water moccasin) show that 45 died in spite of treatment. In most of these cases, the "human" concentrated form was used. * * *

It might be added that the Diamond or Diamond-back rattlesnake, *C. adamanteus*, of the southeastern states is the largest and the most dangerous poisonous reptile of North America. It attains a length of from six to eight feet and the fangs of a six-foot specimen are nearly one inch in length. It injects a large amount of venom. The next largest species is the Texas Dia-

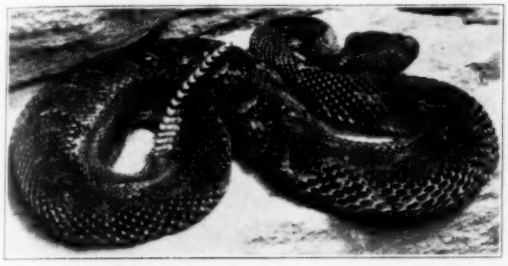


Fig. 3. Timber rattlesnake of the eastern United States. There are both black and yellow phases, the latter with dark, transverse bands.

mond-back rattlesnake, *C. atrox*. Curiously enough, the venom of the smallest of the eastern rattlers, *Sistrurus miliarius*, has the highest toxicity for animals, figuring the poison drop for drop. The venom of another of the smaller rattlers, the Tiger rattlesnake, *C. tigris*, of the southwest, seems to rank next in toxicity.

Determination of Venom Content of Snakes

There has been no satisfactory report of the relative amounts of poison injected by snakes, although the amounts of venom carried in the temporal glands are definitely known through "milking" operations in the laboratory. These amounts range from 50 to 75 mg. for a serpent the size of a copperhead, 100 to 125 mg. for a fair-sized water moccasin, 150 mg. for a big eastern timber or banded rattlesnake, and 250 to 400 mg. for the big diamond rattlers, *C. adamanteus* and *atrox*.

These milking operations consist, first, of controlling and then seizing the snake by the neck and applying its jaws to a glass with parchment tied over the top. As the snake bites and the fangs pierce the parchment, its temporal glands are compressed by the operator's thumb and forefinger. A few more drops are thus obtained than through the furious biting of a snake, during which there is marked contraction of the masseter and temporal muscles over the glands. Sometimes, there is not much difference in the quantity obtained between the initial grasping bite and finger compression of the glands.

It should be understood that an angry snake, held in this manner, will bite more deliberately than one which strikes from a coil. Experiments tend to show that the glands are but moderately emptied at a natural stroke. Apparently, only about half the amount obtained in the "milking" process is injected with a bite.

The natural, striking operation of a poisonous snake is performed in a flash. The head is projected; the jaws are thrown wide open and the fang tips are thrust forward. As the fangs reach the offending object, they are driven in by the force of the stroke and, the instant the object is struck, there is a biting movement, with contraction of muscles against the poison glands, the venom thereby being forced forward and out of the fangs. Hence, the operation is a stroke, aided by a flashing bite; then the head darts back to the serpent's coil.

Antivenin Treatment

Presuming, then, that as much as half the amount of venom obtained by laboratory extraction is administered by a serpent's stroke, and referring to Dr. Githen's figures of 50 mg. neutralizing power of the veterinary antivenin package, it is evident that the smallest amount to be admintered for the bites of the smaller snakes is 50 cc. of the antivenin, and for the big rattlers, at least 200 cc.

As pointed out, the smaller the animal, the relatively larger dose of serum required. An animal the size of an average

dog should receive 100 cc. as an initial dose, with guidance for additional dosage governed by the gravity of the symptoms. If treatment has been delayed, the serum may be administered intravenously. For large animals, where symptoms are not of an alarming nature, an initial dose may be as low as 50 cc., provided that the subject is constantly observed for any increase in the gravity of symptoms, an indication that a large amount of poison has been injected. If such is the case, additional 50 cc. doses should be given within a few hours.

When the size of the serpent and the corresponding amount of venom injected is either unknown or only approximately determined, thorough suction treatment is advised. This will reduce the amount of poison to be neutralized.

Dr. Dudley Jackson, of San Antonio, Texas, has demonstrated that a considerable amount of the poison remains localized in the tissues for hours and sometimes for days. He has shown that a lethal dose for dogs, with no treatment, is 1 mg. of venom to the pound body weight. A subject was injected with venom, much in excess of the lethal dose and, after swelling took place, suction was applied and the bloody serum recovered and injected into four other dogs. Two of these died and the other two suffered such sloughs that they had to be killed. The dog from which the serum was extracted by suction recovered.

A case like this, of course, while highly significant, is different from cases wherein animals that have been bitten while out of doors have wandered for hours before their plight was discovered. In such instances, there has been absorption of poison, and the use of neutralizing serum may save life, although the suction treatment may also be indicated.

PRECAUTIONARY MEASURES DURING TREATMENT

A few attendant points of care should be mentioned. The animal should not be excited or exercised and stimulants should never be administered in the early stages of the trouble. Only in cases of failing pulse should such stimulants as strychnine be utilized. The wound should never be cauterized, as such a procedure is the reverse of proper treatment, which consists of removing the poison and neutralizing what remains, as previously explained. Potassium permanganate, in strong solution, is far more harmful than helpful. In weak solution its action on a wound may be beneficial as an oxidizing agent to reduce bacteria from the mouth of the snake—for highly pathogenic forms may be thus transmitted. As *Clostridium welchii* and *tetani* have been manifest after snake bites, a combined injection as a preventive against

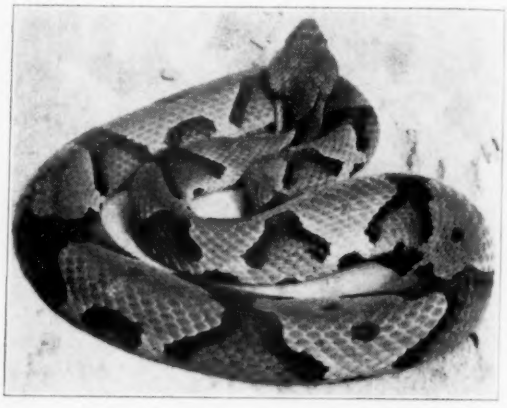


Fig. 4. Copperhead snake — inhabits the eastern United States with western extension into Texas and Oklahoma.

such symptoms may be administered, if the bitten stock is of considerable value. Also, with such animals intravenous salt solutions and blood transfusions may counteract cases discovered after marked organic disturbance has developed in destruction of red blood cells—a condition highly manifest in severe cases of snake-bite poisoning. In early discovered cases, and bites on limbs, moderate ligation, as in human medicine, will retard absorption of poison during suction treatment. One effect of the venom is to reduce greatly the resistance to bacterial invasion; hence, it is advisable that wound areas be covered with a mild antiseptic, wet dressings for about a week after marked symptoms have been alleviated.

Casualties from Snake Bites

Examining the available statistics of casualties among domestic animals caused by poisonous snakes, I have picked out several that may be of particular interest. The first is rather an old one, of 1929, before the production of a specific veterinary antivenin. It appeared in the Bulletin of the Antivenin Institute of America, Vol. III, No. 3, in the form of a report submitted by Mr. E. G. Hayward, Manager of the Philmont Ranch, Cimmaron, New Mex. Quoting:

> We often have snake-bitten animals and we have in recent years had several thoroughbred colts bitten by rattlesnakes. This has given us considerable alarm. The hide would slough off the animal where the bite was inflicted, and we would have a hard time getting the colts through. However, we have never lost a colt, nor have we lost cattle or sheep from snake bite, that I know of. A few of our cattle often swell up, showing evidences of snake bite, but in none of these instances have we had any losses.
>
> Antivenin for colts and horses we would be glad to use on some of our thoroughbred race stuff, as this is very valuable, and we would be glad to save ourselves the expense of a long, tedious care which they require after receiving a good hard bite.

Fig. 5. Water moccasin—inhabits the southeastern United States with extension northward to Virginia and westward to the Mississippi Valley.

Colonel Martin L. Cummins, of Fort Sam Houston, Texas, pointed out:

> About 1925, questionnaires were sent out to the country live stock inspectors of Texas for a report on fatalities from snake bite. Around 60 replied and the average loss was about 1 per cent in cattle, sheep and goats. I lost approximately 9 per cent of my caracal-crossed sheep from snake bite near El Paso, about 1924.

Mr. R. Ross Allen, Director of the Florida Reptile Institute, Silver Springs, Fla., stated that there is a company in Florida which insures dogs against snake bite. This recalls an allegation the writer heard recently relating to the insurance of elephants against the bites of the King Cobra. The condition appears probable from actual losses among such working stock, as shown by Rodolphe Meyer de Schauense, in a publication of the Academy of Natural Sciences of Philadelphia, relating to the King Cobra in Siam. The report reads:

> The largest poisonous snake in the world is found quite commonly in Siam; the record specimen for the country is in the neighborhood of 16 feet.
>
> Regarding the power of this formidable reptile, an incident which had just taken place near Chieng Mai in Northern Siam, a short time before I arrived, struck me quite forcibly.
>
> The best trained tusker elephant in the teak forest, belonging to one of the big foreign companies, had died from the bite of a King Cobra. He was working in the forest when one of these brutes bit him on the foot.
>
> At first, I must confess, I rather doubted the story, because to me it seemed impossible for a snake, no matter how large, to pierce the skin of one of these huge beasts. I asked how it was possible and was told that the mortal bites are inflicted either on the end of the trunk, or just at the juncture of the nail and the foot, in both of which places the skin is tender. An elephant is said to die in about three hours after being bitten.
>
> The death of an elephant from a bite of one of these reptiles is not a very unusual occurrence. As a matter of fact the teak companies count on losing two or three elephants a year as a result of the King Cobra. As good trained tuskers are very hard to replace, they are highly valued and command prices between four and five thousand dollars. Any loss caused by snake bite is therefore quite considerable.

Fortunately, the New World, even in the tropics, has no snake quite comparable to the King Cobra, which carries a large amount of quick-acting and powerful neurotoxin and retains its hold when it bites, thus injecting a much larger amount than the fast striking vipers.

While visiting cattle ranches in Panama, the writer heard of a fair number of bites from the Fer-de-lance, *Bothrops atrox*. The general run of the cattle was undersized, and thin from tick infestation. Their

feeding grounds were precarious, as the grass was high and matted. Most of the bites appeared to result fatally or cause such sloughs that the animals were destroyed. There was talk of importing specific serum to neutralize Bothrops poison, for use with the more valuable stock.

Mr. Arthur Greenhall has helped me in gathering the two extracts that follow, one from Costa Rica, by Dr. C. Picado, of San Jose (1931), which is translated from Spanish:

> Being nocturnal, many venomous species are found in repose during the day and do not attack until they are disturbed in their sleep. It is for this reason that, in deer hunts, the dogs are the victims and not the deer, which in passing frighten the snake and leave it alert and prepared to bite whatever animal that comes near it. The majority of the accidents are produced by small or medium snakes that are difficult to see. We ought to conclude that imprudence is the cause of the majority of the accidents.

The second extract relates to bites from the common European Viper, or adder, *Vipera berus*, and is quoted from *The Life History of British Serpents*, by Gerald R. Leighton, 1901, pp. 126-127:

> Many animals perish every year, even in this country, from adder bite, and probably many that are found dead on hills and commons are really the victims of our venomous reptile. The most frequently bitten are dogs, sheep and cattle. Mr. C. N. Rogers informs me that, in Cornwall, dogs are often killed from adder bite and that this is especially the case with hounds. It can be easily understood why this should be the case, as hounds work with their noses close to the ground and would be very apt to run over an adder. Probably some of the cases read of now and then of hounds being lost and found dead are to be accounted for in this way and not always to be put down to willful poisoning by malicious people.
>
> Dogs are generally bitten in the lower part of the leg, just above the foot, or under the jaw. A good many sheep, too, perish annually from adder bite. In their case the bite is generally under the jaw, or, as I saw in one case, on the udder. The hardness of the hoof is a protection to the feet. This also applies to cattle, which are invariably struck under the jaw. A farmer on Garway Hill, Hereford, this summer (1900) lost a young bullock in this way and found the adder lying beside its victim—not an unusual habit of the reptile. The adder measured 24½ inches and was in my possession an hour or two after it slew its last bullock.

PROTECTIVE MEASURES

Surveillance of valuable range stock, isolation corrals for treatment of cases, and careful control of hunting dogs, never allowing them to stray into particularly thick and hazardous places, are part of the solution of losses from snake bites. Remedial measures should be available on stock farms and ranches, or carried by riders. For protection of canines, a complete outfit for treating snake bite may be carried in the pocket of a hunting coat or attached to one's belt.

SUMMARY

Domestic animals, particularly dogs, are frequently bitten by poisonous snakes. The treatment recommended is: (1) suction by simple, inexpensive and specifically designed apparatus which is readily available and (2) neutralization of poison by veterinary antivenin. The administration of antivenin may be intramuscular, or intravenous (the latter in case of rapidly progressive or advanced symptoms) and the amounts of injection should be contingent to the size of the animal (larger amounts for smaller animals) and to the size of the snake and amount of poison injected. The latter point is indicated by the gravity of the symptoms. The combination of both treatments is advisable.

Comment: The JOURNAL is indebted to the New York Zoölogical Park for the many valuable contributions to the literature on wildlife in captivity which have popularized this outstanding exhibition in the scientific circle.

This marvelous American institution has been brought to the foreground in no small measure by the fact that its management and operation have been directed by highly qualified veterinarians, of whom Dr. William Reed Blair, the present director, is an example.

We are thankful to the author of this article for drawing attention to the relation of snake bites to the conservation of farm animals in the manner described in the foregoing pages.—*Ed.*

Hookworm Disease in Dogs*

By J. W. LANDSBERG, *New York, N. Y.*
Warner Institute for Therapeutic Research

The ubiquitous distribution of the dog hookworm, *Ancylostoma caninum*, in canine species throughout the United States and the disease associated with infection by the parasite in some instances make ancylostomiasis an important problem for the small animal practitioner. According to Stiles,[1] the parasite is responsible for the death of 25 to 40 per cent of the puppies born. An idea of the incidence of this parasitism, at least in one section of this country, may be gained from the survey, by Hinman,[2] on 1,315 dogs in New Orleans, who found that 41.5 per cent of the mature animals and 44.4 per cent of the immature ones were infested. Since this canine nematode has been utilized extensively in the study of hookworm problems which are impracticable to carry out in man, the bionomics and host-parasite relationships of *A. caninum*, in its definitive host, the dog, have been studied as comprehensively as those of any canine parasite.

The dog hookworm has been reported frequently in cats and, on more rare occasions, in the intestine of other carnivores (Foster and Cort[3]), as well as from man (Craig and Faust[4]). Scott[5] observed that, when his experimental strain of dog hookworm was put into cats, fewer parasites survived and reached maturity. The same author[6] found a strain of *A. caninum* in the cat which demonstrated reduced infectivity for a young dog; however, after one passage through the dog, it behaved like the canine strain (Scott[7]) in other dogs. Foster and Cort[3] confirmed this observation and found that the strain remained highly infective to dogs for several parasitic generations. These authors give an excellent summary of the experimental work on this phase of the problem. Although this parasite does not have as much importance in the cat as in the dog, the veterinarian must remember that it may be present and that the cat may be a potential source of infection for the canine species.

*Read before the Veterinary Medical Association of New York City, November 9, 1938, and the Veterinary Medical Association of New Jersey, 55th annual meeting, Trenton, January 10-11, 1939.

Description of Ancylostoma Caninum

The canine species of the hookworm has a pinkish-white, cylindrical body with a chitinous cuticle marked by fine, transverse striations. The males range from 10 to 12 mm. in length and have a greatest diameter of 0.4 mm.; the females range from 14 to 16 mm. in length and have a greatest diameter of 0.6 mm. The worms have a wide buccal capsule to accommodate three pairs of ventral teeth (a diagnostic character). The bursa, the male organ of attachment used in copulation, is large and flaring, supported by long, slender rays.

Determining Presence of Parasite in the Host

In dealing with this parasite the first problem which concerns the veterinarian is ascertaining its presence in the canine host. If the animal is heavily parasitized, one may gain some evidence by the pale mucous membranes of the mouth, the bloody, diarrheic feces with shreds of mucus and, in some instances, emaciation. It must be remembered, however, that bloody stools may be encountered in coccidiosis, and Hinman[2] remarks:

> No correlation was noted between the general body condition of the dog and the presence or absence of hookworms in the intestine—with the exception of an occasional emaciated animal.

The symptomatology is given in much greater detail by Hall, Price and Wright.[8] The only positive evidence of infection is the presence of eggs in the feces. The size of the eggs has been described as being 63.8 x 40.4 μ. They must therefore be searched for with the microscope. The

simplest microscopic examination is the fecal smear method, which requires only a small amount of feces mixed with a drop of water on a glass slide. Obviously, the fault of such a method is the difficulty of finding the eggs in slight infections. To overcome this difficulty various methods of concentrating the eggs are employed. A saturated solution of sodium chloride is widely used as a floating agent as well as centrifugation of the fecal preparation (Lane,[9] direct centrifugal floatation). A method of quantitatively determining the egg concentration of a specimen was described by Stoll and Hausheer.[10] The methods in use at present are well described in a number of texts (Craig and Faust[4]; Chandler[11]; Todd and Sanford[12]; Rebrassier[13]).

LIFE CYCLE OF THE PARASITE

Egg Production: The free-living phases of the life history of this parasite are of interest because of their relation not only to the transmission of the disease but also to its control and prevention. The eggs are passed with the feces usually in the two- to eight-cell stage of development. Sarles[14] observed that the daily egg production of a female of *A. caninum* increased during the first month of infection up to about 20,000 to 25,000 eggs per day but, in heavy infections (over 200 worms), this value did not go above 10,000, as the number of worms present affects the egg output. Herrick[15] found that the average egg production increased from 7,000 to 17,000 and McCoy[16] reports 16,000 eggs per day per female. Sarles,[17] as well as Herrick,[15] found that the egg output decreases rapidly after the first month.

Environment: The most favorable environment for the development of the hookworm eggs is light, sandy loam, in a moist, shady place covered with decaying vegetable matter. The eggs do not develop in heavy clay soils or under water. The physical factors influencing the development of the egg and larvae of *A. caninum* have been studied experimentally by McCoy.[18] This investigator found that the optimum temperature for the hatching of the egg and the development of the larvae was around 30° C., the hydrogen-ion concentration from pH 6.0 to 9.4, and that a single egg required approximately 0.0,000,028 cc. of oxygen for development and hatching.

The same author (McCoy[19]) observed that living bacteria (22 species) constitute the essential food consumed by the larvae in developing to the infective stage. At the optimum temperature given by McCoy, most of the eggs hatched in ten to twelve hours into the rhabditiform larvae (about 0.25 mm. in length). It usually required 24 to 48 hours under outdoor temperature variation. The larvae grow to almost twice the original length in about 72 hours after hatching. They then shed their cuticular covering (moult) and enter the second larval stage of development. Growth continues until the larvae are about 0.5 mm. to 0.7 mm. in length (about 4 to 5 days). At this time the esophagus becomes slender and filariform in appearance and a new cuticle is developed. The second moult occurs and the old cuticula may or may not be shed, depending somewhat on the character of the environment. The larvae now have become the nonfeeding, filariform type which is capable of infecting the host.

In this filariform stage the larvae, under suitable conditions of temperature and moisture, remain on the surface or upper layers of the soil; the majority are found within the first half-inch of the surface. They exhibit the propensity of climbing upon any protruding objects such as leaves, sticks, or soil particles, extending their bodies and commonly waving back and forth. In such positions they may be found either singly or in clumps. As long as sufficient moisture is present the larvae retain their position on the surface but, as dryness ensues, they migrate back into the soil.

The migratory activity of the human hookworm larvae has been described in detail by Augustine and others,[20] who found very little lateral migration but observed that they tend to go in a vertical direction. The larvae are attracted by heat and exhibit thigmotropism (reaction to contact with other objects). As a maximum, they may live in the soil about six weeks and may be recovered therefrom by the method

of Baermann[21] modified by Cort, Ackert, Augustine and Payne.[20]

Penetration of the Host: Infection of the definitive host, the dog, may come about in three ways. The larvae may penetrate the skin, they may be ingested with food and water, or prenatal infection may occur. The larvae, in contact with a warm, resistant surface, display increased activity, consisting in the main of corkscrew-like, penetrating movements, by which they enter the skin. Usually the hair follicles serve as the port of entry but any portion of the skin may be penetrated. For actual penetration the larvae require something against which they can push, something to give them "purchase." It has been observed that mud or the surface tension of drops of water on the skin will give the proper support. Mouth infection is probably the most common type for dogs because of their food habits. Although the subject of prenatal infection with hookworms was considered earlier by Cort,[22] it was not until eleven years later that Foster[23] demonstrated experimentally its occurrence with *A. caninum*.

Migration: Looss,[24] working with the human hookworm, was the first to discover skin penetration and, later (Looss[25]) described the migration of the larvae within the host. During the skin penetration, the sheath is lost and the larvae find their way into the superficial lymph or blood capillaries. Having entered the venous circulation, they are carried to the right side of the heart and out through the pulmonary arteries to the lungs. The lung capillaries tend to hold back the larvae which, in turn, resume their wriggling more actively and bore their way out into the alveoli, then get into the bronchioles, go up the trachea, reach the mouth, move down the esophagus and, finally, localize in the intestinal tract. After localization, they undergo a third moult within four or five days; the fourth or last moult occurs about the 13th day and the worm now has adult characteristics. The feces are positive for ova usually within 14 to 16 days after infection.

The migration of the larvae of *A. caninum*, in some instances, is important in the treatment and should be kept in mind. Not all the larvae are able to penetrate the skin with the same velocity and some of them, during the lung migration, must get back to the pulmonary venules and are thus carried around in the circulation again before eventually reaching the lungs. For these reasons and perhaps others, the entire infection does not localize in the gut at the same time. An animal, therefore, may be treated until the stool is negative upon microscopic examination, and yet, within a week, fecal evidence of the infection may be apparent again. This is true particularly if the infection is quite large. Cognizance of this fact upon the part of the practitioner, manifested by either an explanation of it to the animal's owner or reëxamination of the animal after one week, may prevent embarrassment.

When infection by mouth occurs, the larvae do not usually go through the lung migration but develop directly in the intestine. The fact was demonstrated by Foster and Cross[26] by utilizing an esophageal fistula. It has been observed by various investigators, in carefully controlled experiments, that fewer worms reach maturity following cutaneous infections than from oral administration. This may be explained by the fact that some larvae are lost on the skin and some by exposure to the hazards associated with penetration and migration.

Foster[23] suggested that the most likely route of the larvae in prenatal infection is transportation from the maternal circulation to the fetus. It is curious, as pointed out by Foster, that the development of the larvae is not initiated until the birth of the puppies and that the larvae remain in the organs until parturition. The same author observed that prenatal infection has serious consequences, since all the puppies died within 25 days after birth with a typical picture of acute, fatal hookworm disease.

PATHOLOGY

Although the most striking pathology is that produced in the small intestine by the activity of the parasite (to be considered later), there is also some morbidity asso-

ciated with the migration of the worms during the course of infection. The penetration of the skin produces a local reaction, which was studied by Sarles[27] in young and old dogs. This author observed that young dogs showed no more than a transitory inflammation after larval penetration, while in old animals a marked edema and inflammation appeared immediately and continued for at least a week, with pronounced exudation and necrosis at the center of the lesion. When large numbers of larvae penetrate the skin, there may be destruction of the epidermis with hemorrhage, exudation and the formation of scabs.

Petechial hemorrhages are found in the lungs as a result of the migratory journey of the larvae from the pulmonary capillaries into the air sacs. When large numbers pass through at a single interval, the damage may be extensive with an accumulation of blood in the terminal air cells, which may result in embarrassed respiration.

Verminous pneumonia has been produced in experimental laboratory animals (abnormal hosts, Kerr[28]), other than the dog, with the larvae of *A. caninum*. The incidence of respiratory disorders associated with natural hookworm infection in dogs is probably very low for, as a rule, the infections are light and the predominate mode, oral infection, does not require lung migration for development.

By far the most extensive and serious morbidity from *A. caninum* is produced by the attachment of the parasites to the mucosa of the intestine and the associated anemia. The worms maintain themselves in the intestine by grasping a portion of the mucous membrane with their powerful mouth parts. A portion of the mucosa is drawn into the mouth and subsequently torn away completely as the worms migrate from place to place in the gut as they feed. Following each shift in location, there remain small, bleeding, necrotic areas. In very heavy experimental infections, which terminated fatally, the author has observed the entire lower third of the small intestine completely denuded, entirely hemorrhagic and necrotic as a result of "feeding" by the worms. These denuded areas left by the worms become foci of secondary bacterial infections and have been described for human cases by Smith[29] as "ulcerative, pyogenic lesions."

HOOKWORM DISEASE IN MAN

Since the anemia has occupied such a dominant position in the symptomatology of the human form of this disease, much speculation concerning its origin and many postulations as to its mechanism have been made. The possibility of a toxin produced by the worm being the etiologic agent in human infection was suggested as early as 1879 and, later, in 1888, it was postulated that the toxic action was one of hemolysis. Whipple[30] attributed the anemia to the inflammation of the mucosa and submucosa caused by bites of the parasites which allowed the entrance of intestinal bacteria described previously by Smith.[29] DeLangen,[31] from his studies on man, thought that the anemia of hookworm disease was caused by the activity of a toxin on the bone marrow producing an aplastic condition and depressing the synthesis of hemoglobin. As old as the other theories and as firmly supported was the postulation that blood loss was the cause of the anemia. It may be pointed out that the same theories were advanced to account for the anemia observed in canines as a result of infection with this parasite.

The Anemia: The actual demonstration of the blood-sucking activity of *A. caninum, in situ,* in the gut of the dog was made by Wells[32] and confirmed in all important respects by Nishi.[33] Wells estimated that a single worm may withdraw 0.8 cc. of blood from its host in 24 hours, while Nishi gives the figure of 0.36 cc. for the same period. These facts are corroborated by the amounts of blood in the stool. Fülleborne and Kikuth[34] observed a fall in hemoglobin after the blood appeared in the feces. Two weeks after giving heavy infections, these authors observed that the animals became very anemic and the feces, by chemical analysis, showed enough blood to account for the anemia. The author[35] has observed in fatally infected animals great quantities of

blood in the stools before death and in the lumen of the intestine at the autopsy.

The blood picture following hookworm infection in dogs is characteristically that associated with blood loss. It has been pointed out by Foster and Landsberg[36] that the anemia of nonfatal hookworm infection is typically the "microcytic hypochromic" type associated clinically with chronic hemorrhage. In this type of anemia (chronic post hemorrhagic) the majority of the erythrocytes are small and pale with a greater decrease in hemoglobin concentration and the volume of erythrocytes than in the number of red cells. In the stained smear are few corpuscles of normal size, microcytosis predominates and anisocytosis and poikilocytosis are also present. There is also some evidence of hematopoietic regeneration, as shown by the presence of nucleated erythrocytes and polychromatophilia.

The anemia associated with acute, fatal hookworm infections is somewhat of a different character.[35] There is a rapid and progressive lowering of the erythrocyte and hemoglobin values, which reach a minimum at the time of death. There is not, however, a great change in the size of the erythrocyte nor in the concentration of hemoglobin. The hematopoietic changes observed in these animals are essentially those resulting from severe and rapid blood loss, which has been described clinically as acute, post hemorrhagic anemia.

During the onset of the acute anemia there was observed a marked increase in the percentage of reticulocytes, which may be responsible for a slight increase in erythrocyte volume, since Wintrobe[37] reports that the reticulocyte has an increased corpuscular volume. In the stained smear, several days previous to and at the time of death, were found nucleated erythrocytes, polychromatophilia and some degree of anisocytosis.

It has been pointed out by Landsberg and Foster[38] that a marked leukocytosis did not occur after every infection with the dog hookworm and that the route or magnitude of the infection did not influence the leukocyte response. When a leukocytosis did occur, however, it was due apparently to a rise in the mature neutrophilic cells, the polymorphonuclear neutrophiles, and a "shift to the left" (exhibited by increased myelocytes) did not occur. An eosinophilia did not follow every infection and, when present, occurred at varying intervals after exposure to infection.

Yamaguchi[39] reports an increase in eosinophiles on the seventh day following oral infection and from six to nine days following cutaneous infection. Sarles[40] observed that the increase in eosinophiles coincided with the time at which the hookworm reached maturity (14 to 17 days after infection). This same author, as well as Landsberg and Foster,[38] found that in young dogs (under six months) infection, whether cutaneous or oral, apparently does not produce a marked eosinophilia. Although Yamaguchi[39] reported a higher eosinophilia following oral infection, the authors cited above found that there is little difference in the magnitude of the eosinophilia, irrespective of the mode of administration of the infection. Lymphocytopenia was present in some cases but was not a constant occurrence. There is no appreciable change in the basophiles or monocytes. The number of blood platelets in the peripheral circulation was not significantly changed following infection with *A. caninum* (Landsberg[41]).

BLOOD LOSS BELIEVED CAUSE OF ANEMIA IN HOOKWORM DISEASE

It is the opinion of the author that the detailed study of the blood supports in a great measure the postulation that the anemia is due to blood loss. The same type of anemia as that associated with either fatal or nonfatal hookworm disease could be reproduced experimentally by simple hemorrhage, varying only the quantity of blood drawn and the time interval (Foster and Landsberg[36]; Landsberg[35]).

The marked reticulocytosis, which occurred not only after the first infection but also after the second, as well as other evi-

dence of bone marrow activities, *i.e.*, nucleated erythrocytes, polychromatophilia, militates against the view that the anemia is caused by the inhibition of the bone marrow by the action of a toxin. The fact that the blood platelets are not reduced, as is the case in conditions in which the bone marrow is aplastic, is additional evidence also in favor of the blood-loss postulation. Finally, further evidence is given by the response of the anemia animal to iron therapy (which will be considered later). Although the influence of a toxin *per se* can not be ruled out easily by experimental procedure, it is the author's impression that the rôle of a toxin in the production of hookworm anemia is quite a minor one, if it exists at all.

Effect of Treatment with Iron Salts

The effect of the administration of iron salts upon animals rendered anemic by experimental infections and in those in which the anemia was developing has been studied (Foster and Landsberg[36]). This metal, in the form of iron and ammonium citrate, was given by mouth daily in doses of 2.2 grams for a period of 14 days. A reticulocytosis, which followed the administration of this therapeutic agent (three days), in turn preceded an increase in the number of erythrocytes, the concentration of hemoglobin with a concomitant return of the corpuscular volume to normal. Other animals were given iron salts daily two weeks before and during the period of infection. These animals, so treated, developed some anemia but, when compared with their untreated litter mates, showed an anemia of much less magnitude.

The reaction of these animals to iron salts contributes additional evidence to an explanation of the etiology of the anemia. If the bone marrow were paralyzed by the activity of a myelotoxin to such an extent that a profound anemia resulted, then it is entirely feasible to assume that such a hematopoietic response could not have come about. However, if one takes the view that the heavy blood loss robs the body of its iron so that there is not sufficient material with which to carry out the normal synthesis of the blood, then the response of the hematopoietic system to the iron therapy is explained adequately.

The Two Types of Hookworm Disease

In practice, the veterinarian may see two types of hookworm infection: the nonfatal type, from which the animal recovers, and the fatal type, to which the animal succumbs, the difference depending primarily upon the magnitude of the infection. The chronic type of hookworm disease, with a low grade anemia over a long time interval, so characteristic of human infections, has not been reproduced experimentally in dogs. In the nonfatal type there is a gradual reduction in the erythrocytes and hemoglobin along with an increase in reticulocytes (evidence of hematopoiesis) during the first month of infection.

Concomitantly with this erythrocyte decline, there is an increase in the egg concentration in the feces. Four or six weeks after infection, the erythrocyte and hemoglobin values exhibit their maximum decline and begin to increase very gradually. It requires about six weeks (longest experimental time, 96 days) for these values to return to the vicinity of the normal range. During the return to normal, the egg concentration of the feces, as well as the percentage of reticulocytes in the peripheral blood stream, gradually decreases. At autopsy, after this period, few adult parasites are recovered from the intestine.

The first stages of the acute, fatal type of canine hookworm disease follow the same course as the nonfatal type, but the malady is more rapid and drastic. The removal of the blood by the sucking activity of the worms, however, is so rapid that the hematopoietic centers, in spite of a high reticulocytosis (Landsberg[35]), cannot compensate rapidly enough and the animal dies from anemia. The author has observed that, when the hemoglobin concentration becomes less than 3.0 gm. per 100 cc. of blood (17.4 per cent Sahli), the prognosis is poor and the animal usually dies.

Young Dogs More Susceptible

It has been pointed out by a number of investigators that young, immature dogs are more susceptible to hookworm infection than adult animals. An infection which would be tolerated well by an adult usually produces drastic effects in the younger animal, in many instances causing death. The reason for this phenomenon has not been explained adequately and is probably a combination of factors. It is known that young dogs have fewer erythrocytes and a lower hemoglobin concentration than adults (Landsberg, unpublished report) and, since hookworm infection produces an anemia, this parasitic anemia superimposed upon a natural anemic state may account for the drastic results. Another factor undoubtedly is the rôle played by either age or acquired immunity.

Which type of immunity is functional (or whether it is a combination of both) has long been debated and it is not the purpose of this communication to enter the controversy. However, in view of some very recent work (Otto, Kerr and Landsberg[42]; Otto[43]) which is in accord with studies on other nematode immunity, previous infection does confer a degree of immunity (although not absolute) to subsequent infection. It becomes obvious that the older dog has had a greater opportunity to acquire this protection.

Diet as a Factor

That diet plays an important rôle in susceptibility to hookworm infection was shown by Foster and Cort,[44] who found that the parasites in an animal on a generally deficient diet showed increased rates of development and egg production. It was observed also that malnutrition lowered the resistance (in resistant animals) and that, when infected animals on a deficient diet were transferred to an adequate one, the return of the resistance was indicated by a spontaneous loss of worms. In studies of three dogs and five cats, Foster[45] demonstrated that these animals became distinctly more susceptible to hookworms after being maintained for several weeks on a milk diet. The brief citation of the above work is a sufficient reminder that veterinarians should insist upon an adequate diet for puppies.

Suggestion for Treatment

Since from the author's viewpoint most of the morbidity is brought about primarily by the anemia, obviously the first step in the treatment of the animal is to remove the cause of the anemia, namely, the blood-sucking parasite. The removal of the hookworm from the gut of the canine is not particularly difficult and it has been found that carbon tetrachloride, tetrachlorethylene and hexylresorcinol are effective anthelmintics in the removal of this nematode. The dose of tetrachlorethylene employed is usually about 0.3 cc. per kilo of body weight. For an adequate description of the use of carbon tetrachloride and tetrachlorethylene, contraindications, etc., the reader is referred to the paper of Hall, Price and Wright.[8] The use of hexylresorcinol as an anthelmintic has been described by Lamson and his coworkers.[46]

Immediately following the removal of the worms, the anemia present may be treated with capsules of iron and ammonium citrate. This may be continued until the erythrocyte values return to normal. During the course of iron therapy, it is essential that the animal be maintained on an adequate diet and, due to the gastric irritation produced in some instances by the administration of iron salts, it is advisable to give this treatment after feeding.

Control

The control of this parasite lies in breaking the life cycle. One of the first measures, and probably the easiest, is the removal of the source of infection *i.e.*, the treatment of the infected host by the administration of an anthelmintic. In this connection, it should be pointed out that a good general rule is to determine at once if the animal is infected. If the stool is positive, the animal should be treated before it is allowed to associate with other

dogs. It should be emphasized again that pregnant animals, if positive for hookworm, should be treated before parturition.

The elimination of potential infections from the surroundings (cages, runs, etc.) is more difficult. To prevent infection from indoor environment, such as cages, floors, etc., one need only wash them well with hot water. Water near the boiling temperature, with or without soap, will kill hookworm eggs and larvae. In laboratories carrying on experimental hookworm research, such a procedure once a day successfully combats accidental hookworm infection.

The control of this parasite on the outside, such as in dog runs, is more difficult, particularly since the free-living stages are well adapted to a soil environment. As pointed out previously, the hookworm requires a certain type of soil and moisture concentration as well as a suitable temperature. Effective control may be accomplished by regulating any one or all of these conditions. One of the most efficient measures is to cement the entire run, but such procedure is not always feasible or desirable. Other prophylactic measures for outdoor runs include adequate drainage with plenty of sunlight, the spreading of lime on the surface, and yearly alternation of runs. The last measure may be supplemented by turning under the surface soil. Engle[17] has suggested covering the surface of the run with washed gravel (about the size of a pea). He finds that a three-inch covering over the runs for adult animals gives satisfactory results.

REFERENCES

[1]Stiles, C. W.: Report upon the prevalence and geographic distribution of hookworm disease (uncinariasis or anchylostomiasis) in the United States. U. S. Pub. Health Serv., Hyg. Lab. Bul. No. 10 (1903).

[2]Hinman, E. H., and Baker, D. D.: Helminthological survey of 1,315 dogs from New Orleans with special reference to age-resistance. Jour. Trop. Med. & Hyg., May 1, 1936.

[3]Foster, A. O., and Cort, W. W.: Further studies on the effect of a generally deficient diet upon the resistance of dogs to hookworm infestation. Amer. Jour. Hyg., xxi (1937), pp. 83-93.

[4]Craig, C. F., and Faust, E. C.: Clinical Parasitology. (Lea and Febiger, Philadelphia, 1937.)

[5]Scott, J. A.: An experimental study of the development of *Ancylostoma caninum* in normal and abnormal hosts. Amer. Jour. Hyg., viii (1928), pp. 158-204.

[6]Scott, J. A.: Experimental demonstration of a strain of the dog hookworm, *Ancylostoma caninum*, especially adapted to the cat. Jour. Parasitol., xv (1929), pp. 209-215.

[7]Scott, J. A.: Strains of the dog hookworm, *Ancylostoma caninum*, specific to the dog and to the cat. Sci., lxix (1929), p. 526.

[8]Hall, M. C., Price, E. W., and Wright, W. H.: Parasites and parasitic diseases of dogs. U. S. Dept. Agr., Circ. 338 (Washington, D. C., 1934).

[9]Lane, C.: Hookworm Infection. (Oxford University Press, London, 1932.)

[10]Stoll, N. R., and Hausheer, W. C.: Concerning two options in dilution egg counting: small drop and displacement. Amer. Jour. Hyg., vi (March suppl., 1926), pp. 134-145.

[11]Chandler, A. C.: Hookworm disease. (The Macmillan Co., New York, 1929.)

[12]Todd, J. C., and Sanford, A. H.: Clinical Diagnosis by Laboratory Methods. (W. B. Saunders Co., Philadelphia, 1937.)

[13]Rebrassier, R. E.: Diagnosis of Parasites in Animals. (Ohio State University Press, Columbus, 1938.)

[14]Sarles, M. P.: The effect of age and size of infestation on the egg production of the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., x (1929), pp. 658-666.

[15]Herrick, C. A.: A quantitative study of infections with *Ancylostoma caninum* in dogs. Amer. Jour. Hyg., viii (1928), pp. 125-157.

[16]McCoy, O. R.: The egg production of two physiological strains of the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., xiv (1931), pp. 194-202.

[17]Sarles, M. P.: The length of life and the rate of loss of the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., x (1929), pp. 667-682.

[18]McCoy, O. R.: The influence of temperature, hydrogen-ion concentration, and oxygen tension on the development of the eggs and larvae of the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., xi (1930), pp. 413-448.

[19]McCoy, O. R.: The suitability of various bacteria as food for hookworm larvae. Amer. Jour. Hyg., x (1929), pp. 140-156.

[20]Cort, W. W., Ackert, J. E., Augustine, D. L., and Payne, F. K.: Investigations on the control of hookworm disease. II. The description of an apparatus for isolating infective hookworm larvae from soil. Amer. Jour. Hyg., ii (1922), pp. 1-16.

[21]Baermann, G.: Eine einfache Methode zur Auffindung von Ankylostomum (Nematodem) Larven in Erdproden. Medel. und L. Geneesk. Lab. te Veltevreden, Feestbundel, Batavia (1917), pp. 41-47.

[22]Cort, W. W., Ackert, J. E., Augustine, D. L., and Payne, F. K.: Prenatal infestation with parasitic worms. Jour. Amer. Med. Asso., lxxvi (1921), pp. 170-171.

[23]Foster, A. O.: Prenatal infection with the dog hookworm, *Ancylostoma caninum*. Jour. Parasitol., xix (1932), pp. 112-118.

[24]Looss, A.: Zur Lebensgeschichte des *Ankylostoma duodenale*. Cent. f. Bakt., Abt. I, xxiv (1898), pp. 483-488.

[25]Looss, A.: Die Wanderung der Ankylostomum- und Strongyloiedes Larven von der Haut nach dem Darm. Compt.-rend. 6th Cong. Internat'l de Zool., Geneva (1905), pp. 225-233.

[26]Foster, A. O., and Cross, S. X.: The direct development of hookworms after oral infection. Amer. Jour. Trop. Med., xiv (1934), pp. 565-573.

[27]Sarles, M. P.: The reaction and susceptibility of dogs at different ages to cutaneous infection with the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., x (1929), pp. 683-692.

[28]Kerr, K. B.: Studies on acquired immunity to the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., xxiv (1936), pp. 381-406.

[29]Smith, A. J.: Uncinariasis. Internat'l. Clin., ii (1904), pp. 68-92.

[30]Whipple, G. H.: Uncinariasis in Panama. Amer. Jour. Med. Sci., xxxvii (1909).

[31]DeLangen, C. D.: The origin of the anemia with anchylostomiasis. Dutch. Ind. Med. Gov. Serv., iv. (1922), pp. 304-316.

[32]Wells, H. S.: Observations on the blood-sucking activities of the hookworm, *Ancylostoma caninum*. Jour. Parasitol., xvii (1931), pp. 167-182.

[33]Nishi, M.: Experimental observations on the blood-sucking activities of Ancylostomidae, especially *Ancylostoma caninum*. Jour. Med. Asso. Formosa, xxxii (1933), pp. 677-691.

[34]Fülleborne, F., and Kikuth, W.: Wie ent steht die Anämie bei Haken wurminfektion? Arch. für Schiffs. und Trophyg., xxxiii (1929), pp. 171-188.

[35]Landsberg, J. W.: The reticulocyte response in acute, fatal hookworm anemia. Amer. Jour. Hyg., xxvi (1937), pp. 60-71.

[36]Foster, A. O., and Landsberg, J. W.: The nature and cause of hookworm anemia. Amer. Jour. Hyg., xx (1934), pp. 259-290.

[37]Wintrobe, M. M.: Relation of variations in mean corpuscular volume to number of reticulocytes in pernicious anemia; significance of increased bone marrow activity in determining the mean size of red corpuscles. Jour. Clin. Invest., xiii (1934), p. 669.

[38]Landsberg, J. W., and Foster, A. O.: The white cell picture in hookworm disease of dogs. Amer. Jour. Hyg., xxv (1937), pp. 141-149.

[39]Yamaguchi, M.: Uber das Blutbild bei experimenteller Ankylostomiasis. Trans. Jap. Path. Soc., xviii (1928), pp. 505-510.

[40]Sarles, M. P.: Studies of the blood changes occurring in young and old dogs during cutaneous and oral infection with the dog hookworm, *Ancylostoma caninum*. Amer. Jour. Hyg., x (1929), pp. 693-704.

[41]Landsberg, J. W.: The blood platelets in hookworm anemia. Amer. Jour. Hyg., xxvii (1938), pp. 60-71.

[42]Otto G. F., Kerr, K. B., and Landsberg, J. W.: The immunity to the hookworm, *Ancylostoma caninum*, acquired by dogs as a result of previous infection. Jour. Parasitol., xxiii (1937), p. 560.

[43]Otto, G. F.: Studies on the protective power of serum from dogs actively immunized against *Ancylostoma caninum*. Jour. Parasitol., xxiv (1938), p. 10.

[44]Foster, A. O., and Cort, W. W.: Further studies on the effect of a generally deficient diet upon the resistance of dogs to hookworm infestation. Amer. Jour., Hyg., xxi (1935), pp. 302-318.

[45]Foster, A. O.: On a probable relationship between anemia and susceptibility to hookworm infection. Amer. Jour. Hyg., xxiv (1936), pp. 103-128.

[46]Lamson, P. D., Brown, H. W., and Ward, C. B.: Hexylresorcinol in canine ascariasis. No. Amer. Vet., xii (1931), pp. 12-21.

[47]Engle, J. B.: Personal communication. Summit, N. J., 1939.

Dog-Food Production Soars

From figures collected by the Census Bureau, as of 1937, it appears that the production of dog foods has become a major American industry. According to these figures, more than 500,000 pounds of dog food, valued at $25,000,000, were produced during that year. This total comprises 378,337,397 pounds of canned food, valued at over $18,000,000, and 123,000,000 pounds of other types, valued at over $7,000,000.

Fifteen years ago, the production of canned dog food totaled only a few thousand cases annually. Today, millions of cases are produced each year and many thousands of people are employed either directly or indirectly by the industry.

Recently a three-month-old Pekingese was brought by airplane from Lake Villa, Ill., to an animal hospital in Ottawa, Ill., for an emergency operation.

William Goldbecker, of New Haven, Conn., claimed a world's record for the breed when, on February 9, twelve Dachshund puppies were born to one of his dogs.

Bingo, a toy bulldog owned by Carl Jones, of Elgin, Ill., was pestered by a flea and registered his protest by scratching vigorously. This ignited a match in the pocket of an old sweater on which he was lying and set fire to the house. Firemen saved the house . . . but not poor Bingo's tail.

Skin Diseases Occurring in Both Man and Animals*

By HOWARD FOX, *New York, N. Y.*

The original presentation of this subject was illustrated with lantern slides. The reader, of course, does not have the advantage of this pictorial accompaniment. The text, however, is graphic and reasonably thorough, and every reader interested in skin diseases should profit from it.

I feel greatly honored to be asked to appear before this large body at your Diamond Jubilee. It is my purpose to show you slides of diseases of the skin in man which also occur in animals. I was not aware that I was to talk before the Section on Small Animals. Therefore, some of the diseases of the skin which I will show have their counterpart only in large animals. Most of my theoretical knowledge about diseases of the skin in animals was obtained from Heller's interesting chapter in Jadassohn's *Handbuch*.

(Slide) The first picture is easily recognized by everyone—ordinary hives. I imagine it must be rather difficult to detect the presence of hives in the majority of animals on account of their hair. Possibly where the hair is short or where the hives are greatly elevated, it would be easier to see them. I know of no disease of the skin, or reaction—I prefer to consider this a cutaneous reaction of the skin—which has such innumerable and unrelated causes as urticaria. It is probable that the majority of cases are produced by some disturbance in the gastrointestinal tract.

(Slide) This condition is called dermographism, or writing on the skin. Some people have an irritable skin which, when traumatized or scratched, produces elevations or artificial hives. I believe that on rare occasions this condition has been seen in animals.

(Slide) This is a picture of purpura, which may be either primary (idiopathic) or secondary (symptomatic). We subdivide the primary types into four groups. The first is simple purpura, in which there is merely extravasation of blood into the skin or mucous membranes. A second, and more severe type, is associated with urticaria, erythema multiforme and joint manifestations. We speak of this as purpura rheumatica. A third type, seen in children, has similar changes besides hemorrhage into the bowel and stomach causing colicky pains, vomiting and diarrhea. The fourth type is known as thrombocytopenic purpura. It was formerly called purpura hemorrhagica. This differs from the other three types in two respects. Hemorrhage takes place through the skin, free blood appearing in cavities such as the mouth, stomach, vagina and bladder. Furthermore, as the name would imply, there is a lowering of the number of blood platelets. I understand that this differs in various animals, so the actual number of platelets in a human patient would not mean much to veterinary medicine.

(Slide) Secondary purpura may also be subdivided, due to mechanical causes, toxic causes such as snake bite and infectious diseases. There are several infectious diseases in man that are invariably accompanied by hemorrhage in the skin. A fourth miscellaneous group includes leukemia, tuberculosis, malignancy, etc.

(Slide) In reading Heller's chapter, I was surprised to find that occasionally one sees eruptions in animals due to drugs. In man we see many of them, I am sorry to say. This is an iodide rash. I think some writer has stated that iodides may cause a scaly patch, as in animals. This is the type that appears commonly in man as a papulopustular eruption. It is rather slow to appear and disappear. I do not know whether bromide eruptions occur in animals. Both bromoderma and ioderma, like all drug eruptions, disappear when the drug is stop-

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

ped. Bromides can be eliminated from the body by the use of common salt. In severe cases we give intravenous injections of physiological salt solution.

(Slide) This is another case of iodide eruption in a woman who took 150 grains of iodides three times a day for six months. This eruption suggests tuberculosis or blastomycosis.

(Slide) Here is a third type of iodide eruption, a bullous type. This child took only a small amount of iodides but was specially sensitive to the drug.

(Slide) Several pictures will now be shown representing a disease that I know is common in certain animals, namely eczema. I consider eczema as a special type of reaction, probably due to sensitization to one or more agents which may act internally or externally. However, it is extremely difficult in the average case of eczema to find this sensitizing agent. I do not know whether you gentlemen find it easy to discover the cause of eczema in animals. I imagine you have your troubles just as we do.

Eczema in man produces a certain chain of symptoms, including redness, itching, moisture, scaling, crusting, thickening of the skin and a tendency to form patches which fade off gradually into the normal skin. If there is no itching, it cannot be eczema. Oozing, or tendency to ooze, is always present. Eventually, there are scaling and crusting and, when the process lasts a long while, the skin becomes thickened and hyperpigmented. Unlike eczema in animals, loss of hair is not a feature of the disease. This is an illustration of acute eczema which is dry for the moment. A colored picture would show the skin to be fiery red.

(Slide) Here is another picture of eczema in which the vesicles and papulopustules have dried to form these crusts—a subacute type.

(Slide) Here is a patch of eczema that is distinctly chronic. The skin is thickened and it would be unnecessary to ask the patient whether he or she used the fingernails to produce the linear excoriations. It is obvious that the skin has been severely scratched.

I might say a word or two about the treatment in general of eczema. For an acute process, with edema and a good deal of oozing and itching, we use cold compresses of boric acid or aluminum acetate. I use boric acid a good deal because it is soothing, cheap and always available.

In the acute stages we also use the calamine and zinc lotion, the calamine (impure zinc carbonate) being used because it is flesh colored. The basis of the lotion is lime water, and there is hardly anything that is as soothing to the human skin as pure lime water. Zinc oxide, in a shake lotion of lime water, makes an excellent soothing remedy for the early stages of an eczematous process or almost any other kind of acutely inflamed skin.

In the subacute stages we use a paste of zinc oxide and starch. Eventually, when the skin becomes dry and thickened, we need some stimulating remedy, such as wood tar. The best of the three types of wood tar is juniper tar or oil of cade. About 10 per cent oil of cade in vaseline constitutes a valuable antipruritic remedy. I have used this for years in hospital and private practice. It is cheap, simple and effective. It is obvious to everyone that animals, especially dogs, suffer from pruritus. I do not know whether you have the ideal antipruritic now or not.

(Slide) I know that certain cutaneous lesions in animals are made worse by constant licking. Here is a case in a human being of an eczematous process that is due to licking. This little girl had the nervous habit of licking her lips with her tongue and produced this eczematous area by doing so.

I might say a few words about the treatment of chronic eczema or any thickly infiltrated patch of skin due to eczema or other cause. Chrysarobin in ointment form, 10 to 20 per cent, is a valuable remedy for such conditions. If you want to get the strongest action from chrysarobin, apply the ointment and cover it with an impermeable substance, such as oiled silk or gutta

percha. This doubles or trebles the action of the salve.

(Slide) This picture shows an ordinary herpes or cold sore. A single patch of herpes is of little importance. When lesions recur three or four times a year for ten or 20 years, it becomes a real problem. As you know, herpes is due to a virus and it has been found that in some obstinate cases the giving of one virus stops the progress of another. We vaccinate the patient with smallpox virus once a month for four successive months and it does not seem to make any difference whether the vaccination "takes" or not. In a reasonable number of cases this prevents the recurrence of herpes.

(Slide) This is a picture of pemphigus, an essentially bullous eruption. I do not know whether there is anything comparable to true pemphigus in animals, because no one knows exactly what pemphigus is. The cause has not yet been discovered. Recent researches suggest a virus. Pemphigoid eruptions occur in some animals. I have seen a striking illustration of such an eruption in a horse. For pemphigus in man, which in adults, at least, is almost invariably fatal, we have not found any satisfactory remedy.

(Slide) Here is a picture of erysipelas. Facial erysipelas is ordinarily a mild disease and, for some unknown reason, usually stops at the hair line, seldom involving the scalp. Until recently the disease was treated, in Bellevue Hospital, by anti-streptococcus serum. This has lately been replaced by sulfanilamide. It shortens the course of the disease and greatly lessens the mortality. Within 48 hours the temperature usually becomes normal and the patient is on the road to recovery.

(Slide) Here is a case of elephantiasis or solid edema of the face, probably due to low-grade streptococcic infection. Personally, I have never seen any favorable results from the treatment of this condition.

(Slide) Here is a case of severe elephantiasis which has nothing to do with filariasis. It occurs in people who do not live in endemic regions, such as Charleston, South Carolina or the Virgin Islands, where filariasis is endemic. It is due to obstruction of the lymphatics, probably caused by erysipelatous attacks.

(Slide) Here is another case of streptococcic infection—ordinary ecthyma. The disease begins as vesicles, which quickly rupture, form crusts and become secondarily infected. An individual lesion runs its course in a month or so but, if not treated properly, the process may continue for a long time. This type of eruption is seen frequently in the vagabond type of individual and responds readily to ordinary parasiticidal remedies such as ammoniated mercury. This drug is frequently used for pyogenic infections, although some persons are sensitive to it. Mercurial preparations should never be used immediately before or after an application of iodine, as the resulting mercuric iodide is most irritating to the skin.

(Slide) This photograph illustrates what everybody will recognize as an ordinary furuncle, caused almost invariably by the *Staphylococcus aureus* (or, occasionally *S. citreus*) but never by the *S. albus*. We hear a great deal about diabetes and hyperglycemia as causes of boils. As a matter of fact, the majority of cases of furunculosis occur in persons whose blood sugar is normal and who have never shown any sign of diabetes. Though boils are caused by staphylococci, trauma and moisture are at times contributory causes. I do not know whether vaccines are of value in veterinary practice for the treatment of furunculosis. In my opinion, they are of distinct value in man. Stock vaccines are usually as efficacious as autogenous ones. They are best given at intervals of five days, the dosage being small at the outset and gradually increased. If new boils appear, the dosage should be reduced.

(Slide) This is ringworm. It is hardly necessary to say that it is a contagious disease, since the entire family is affected. Whether it is an infection with microsporon or trichophyton, I do not know. The picture was taken by my father, many years ago, before we became interested in studying the different types of ringworm by cultures.

(Slide) This is another type of ringworm which affects the surface of the skin (horny layer of the epidermis). It makes a great deal of difference in man whether ringworm is on the surface or penetrates into the hair follicles and produces folliculitis. This superficial type is easy to treat. An ointment containing 10 per cent iodine crystals in vaseline would clear this eruption rapidly.

(Slide) Here is a case of trichophyton infection. The violaceum species caused this folliculitis. The fungus gets deep into the hair follicles and causes a pyogenic reaction. Some of the ringworm fungi are just as able to produce pus as are the tubercle bacilli.

(Slide) This is a picture of the ordinary small-spored ringworm, due to one of the microsporons. *Microsporon audouini* is the human type and *Microsporon lanosum* the animal type of this fungus.

(Slide) Ringworm of the scalp was a big problem until Sabouraud showed that it could be cured by x-rays. This is the agent we constantly use at the present time. Thallium acetate is also an effective remedy, but it is a dangerous drug unless the exact dosage is given. It is improper, I think, to subject a child to the danger of death by treating an essentially harmless disease.

(Slide) This is a picture of a disease that we do not see as frequently as you gentlemen do—actinomycosis. In man it is often fairly harmless. It does not become systemic in the majority of cases and it yields satisfactorily to large doses of iodides and to local x-ray treatment. It is not always easy to confirm the diagnosis, *e.g.*, it may be difficult to find the ray fungus in the pus from discharging sinuses.

(Slide) Here is a picture of favus, which causes atrophy of the scalp and permanent baldness.

(Slide) This is a rare picture of favus of the glabrous skin and an unusually extensive eruption.

(Slide) Here is a photomicrograph of the microsporon in a hair. Focusing up and down on the slide would probably show 15 or 20 layers of these small spores crowded together in mosaic-like form. They are true spores.

(Slides) This is a typical picture of a hair infected by a trichophyton. In the cortex of the hair you see so-called sporulated mycelium looking like little rosaries. It is the vegetative portion of the fungus.

(Slide) Here is a photomicrograph of favus. The amount of the mycelia, (the vegetative part of the fungus) in the favus hair is comparatively slight compared with the ringworm hair. That is why it is possible to cure the disease by epilation. In ringworm that can not be done because the hairs break off too easily.

(Slide) This is a microscopic preparation of a trichophyton found in the glabrous skin.

(Slide) This is a culture of the *Microsporon audouini*, the cause of the human type of ringworm of the scalp.

(Slide) This is a picture of human scabies. Ordinarily it is easy in a well marked case of scabies to make the diagnosis. It may be difficult in adults who bathe constantly and thus remove a large number of the acari. The diagnosis in children, especially those who are neglected, is easy, the lesions occurring on parts of the body where the skin is thin. In babies or young children it is often possible to see the burrow made by the female acarus, which appears as a little, gray, wavy line, perhaps a quarter of an inch long. In the average person, on account of intense itching, the burrow is removed by scratching. We usually make the diagnosis by the location of the excoriated papules, the history of nocturnal itching, and the presence of the disease in other members of the family. We never find the acari (Sarcoptes) on the surface, as in animals. The male is smaller than the female and is rarely seen, and the female is barely visible to the naked eye.

(Slide) Here is a severe case of scabies with a great deal of pustulation. Sulfur in some form is still our mainstay for the treatment of this disease.

(Slide) This is a picture of the *Demodex folliculorum* originally discovered in the wax glands of the ear by Henley. It

is generally thought that the *Demodex folliculorum* in man does no harm. There are, however, occasional cases of pigmentation of the face that have been ascribed to the presence of this mite, and rosacea is ascribed by one of my colleagues to the presence of these parasites. The mites can be found most easily in the large follicles about the nose of adults, especially those with a greasy skin.

(Slide) Except for cattle, animals apparently do not suffer from tuberculosis to the same extent as man. This slide shows the commonest type of tuberculosis of the skin known as lupus vulgaris. It is more or less encapsulated but occasionally the bacilli get into the general circulation and cause generalized or pulmonary tuberculosis.

(Slide) This is a case of tuberculous ulcer of the lip, which is always secondary to pulmonary tuberculosis.

(Slide) This is tuberculosis verrucosa cutis, which is supposed to be due to infection by the bovine type of the tubercle bacillus. It is prevalent among those who handle infected carcasses. I am sure some of my audience are familiar with this type of eruption.

(Slide) This is another type of tuberculosis of the skin which we speak of as scrofuloderma, meaning tuberculosis of the skin overlying a tuberculous focus in the lymphatic glands or bones.

(Slide) Here is a common, everyday wart. It disappeared under treatment by x-rays although this agent is not usually successful in treating verruca vulgaris. In my experience, the best way to get rid of ordinary warts is to freeze them with ethyl chloride and remove them by a sharp bone curette. It is surprising how often this can be done without leaving a scar.

(Slide) This shows that common warts can invade the mucosa as well as the skin.

(Slide) This is a case of molluscum contagiosum. It is another virus disease and, like common warts, disappears spontaneously after a number of months or years. Why these lesions come and go like that, we do not know. In human beings, it has been proven that various types of warts may be made to disappear by mental suggestion. This has been scientifically proven by Bloch. It seems hardly possible that mental suggestion can cause an organic lesion, known to be due to a living virus, to disappear, but it is apparently true.

(Slide) This is a queer kind of lesion which is occasionally seen in human beings, namely a cutaneous horn. Much larger ones occur in animals. This is a true cutaneous horn that arises from the horny layer of the epidermis. A lesion of this kind is usually small but potentially dangerous. Occasionally it drops off and grows again, and the base may become inflamed and be the starting point of epithelioma. There are one or two cases on record of cutaneous horns in human beings that were one foot in length and one inch in diameter. Unlike animals, cutaneous horns in man seldom arise from the skin of the ears.

(Slide) This an ordinary fibroma. Most of them are sessile. Occasionally, they are pedunculated like this one.

(Slide) Here is a diffuse lipoma. A few of these cases are painful, as in Dercum's disease, but are otherwise harmless.

(Slide) Here is a neuroma. This type of lesion is seen also in Von Recklinghausen's disease, which I do not believe has been recognized in animals. They do suffer however, from neuromas.

(Slide) Here is an ordinary hypertrophic scar from a burn. Like some types of tuberculosis, a burn produces a keloidal hypertrophic and deforming type of scar. If the scar is treated early, irradiation by x-rays or gamma rays of radium will flatten it materially.

(Slide) Here is a picture of ichthyosis of the most severe type that is compatible with life. At times people like this get into the circus, the famous alligator boy or the manfish of Tennessee, for example. I have seen photographs of some animals that showed this extreme type of congenital abnormality.

(Slide) Now we have a few photographs of malignant changes. We distinguish sharply in man between a basal-cell and a

squamous-cell epithelioma. The basal-cell type is only locally malignant. It destroys tissues and even bone, but practically never metastasizes as does the squamous-cell type.

(Slide) Here is an epithelioma of the hand. When seen on the extremities, it is nearly always of the squamous-cell type. The man died two years later. The cancer appeared about a year after he was bitten by a horse.

(Slide) Here is a round-cell carcoma. The patient died seven months after the removal of this extremely malignant type of lesion.

(Slide) This little girl shows a lymphosarcoma. I know that dogs suffer from lymphosarcoma, as I lost one from this disease.

(Slide) This photograph, taken one week after x-ray treatment, shows complete disappearance of the lymphosarcoma, proving it to be highly radiosensitive. Unfortunately, it recurred and the child died six months later.

(Slide) Here is a malignant sarcoma, the most malignant neoplasm with which we have to deal.

(Slide) This picture looks like a harmless eruption. You can see a disfiguring scar, resulting from the removal of a nevus, and you also see numerous black dots representing metastases from that very malignant melanoma.

(Slide) This is a type of baldness that we do not often see—a congenitally poor growth of hair. Nothing can be done for it.

(Slide) Here is a photomicrograph of a hair showing *Trichorrhexis nodosa*. I understand that certain animals suffer from the same affection which gives the fur a tousled appearance. We do not know the cause of the disease. The individual nodes are seen in the long hairs of women and the beards of men. Clinically you see them as tiny nodes and under the microscope they look like shaving brushes placed end to end. Some think the disease is due to lack of oil, while others consider it to be an infection. I do not know any way of curing it.

(Slide) Here is another disease of the scalp, alopecia areata, that was first described by Celsus in 25 A. D. I am sorry to say we do not know any more about this disease than Celsus did. We surmise that it may be due to endocrine disturbance, but that is as far as we can go. The baldness varies from one small spot to the loss of every hair on the body.

(Slide) This shows loss of even eyebrows and eyelashes. Some lose the pubic, axillary and downy hair all over the body in which cases, the prognosis is bad. When only a certain number of patches are present, the prognosis is usually good. In treating these lesions, I think that local stimulation with quartz lamps (or pure phenol for small spots) helps bring back the growth of hair.

(Slide) This is a peculiar case of permanent pigmentation. Everybody is familiar with ephelides, the ordinary sun freckles that small boys and girls have in the summer. These are permanent freckles, however, which are hard to remove.

(Slide) This ram's horn twist in the nails is known as onychogryphosis. In the diagnosis and treatment of nail diseases we have our difficulties. In Heller's monograph I saw a striking picture of onychogryphosis in a wild animal (a deer) and I also read in the text that certain animals, like lions and tigers, which are confined to cages, also acquire this disease.

Dachshund

The dachshund is a native of Germany. It derives its name from being used to hunt dachs, a German animal similar to the American badger.

In temperament, conformation of the head and in uses, it is entirely terrier. The breed should be classed as a terrier, not as a hound.

The belief is that it develops fewer ailments than some other hunting breeds, for it shows rare intelligence in protecting itself while hunting. It takes good care of itself in a fight. The skin is so loose that it allows the dog to turn and bite its opponent.

Some Diseases of the Eyes of Lower Animals: Methods of Examination, Diagnosis and Treatment*

By CARL F. SCHLOTTHAUER, *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Foundation

GENERAL CONSIDERATIONS

Ophthalmology is concerned with the eyeball, orbit, eyelids, muscles of the eyes and the lacrimal apparatus. Various defects and diseases of these organs and structures have been observed in lower animals. Their causes are various and the symptoms may vary from a mere cosmetic effect to total blindness. Veterinary ophthalmology is concerned chiefly with those defects and diseases that are manifested by macroscopic lesions. Defects of vision due to obscure causes frequently are difficult to recognize and usually are of no importance because domesticated animals do not require such acute vision as man. Indeed, it sometimes is difficult to detect unilateral blindness.

For convenience, the diseases of the eyes and their appendages may be grossly grouped into two large classes according to their cause, as noninfectious and infectious diseases. The former group is the larger of the two and in it are included hereditary defects, neoplasms and lesions due to trauma, chemicals, biologic toxins, certain deficiencies of the diet, and circulatory and systemic disorders. A deficiency of vitamin A, however, can cause night blindness without the development of destructive lesions.

The diagnosis of some diseases of the eyes may be difficult even when macroscopic lesions are present. Therefore a knowledge of the histologic structure of the eyeball and its method of functioning is important to facilitate examination of this organ, to make a correct diagnosis and to prescribe treatment.

The eyeball is the essential organ of vision. It is a hollow globular organ composed of fibrous membranes, vascular tissues, nervous tissues and transparent media. Physically it resembles a camera. The curved surfaces of the cornea, the lens and the soft media constitute a refracting apparatus which directs the rays of light entering the eye onto the retina. The retina acts as a screen and the image is formed on it. The iris functions as the diaphragm opening in the shutter of a camera and governs the amount of light that may enter the fundus of the eye. The fibrous sclera and cornea form the box to enclose this mechanism. The eyelids can act as a camera shutter, but their chief function is that of protection.

Errors in vision commonly are due to errors in refraction. Refraction in an eye is measured and expressed in diopters. A diopter can be defined as the refractive power of a lens having a focal length of one meter. Errors in refraction commonly are determined by the use of special lenses. These may be mounted in an opthalmoscope in a series of plus and minus lenses of one or more diopters each. Such an ophthalmoscope is a convenient instrument for general use. One may also employ other methods to test an eye for an error in refraction. The so-called shadow test is perhaps the most frequent other test employed. However, to avoid confusion I shall not discuss it.

A normal eye is emmetropic. In it the light rays entering the fundus are refracted so that they unite directly on the retina. In a myopic eye the light rays unite and form the image in front of the retina, while in a hypermetropic eye the light rays unite and form the image behind

*Read before the Southern States Veterinary Medical Association, Knoxville, Tenn., Nov. 3-5, 1938, and also before the Veterinary Medical Association of New Jersey, Trenton, January 10-11, 1939.

the retina. Astigmatism is due to irregularities in the curvature of the cornea.

Myopia is measured with and can be corrected by the use of a biconcave lens. In the ophthalmoscope these lenses are designated with a minus sign. Hypermetropia, on the other hand, is measured with and corrected by the use of a biconvex lens, which is designated by a plus sign in the ophthalmoscope. Myopia is common in many animals. Dogs frequently have a refractive error of 3 or 4 diopters. Because of the frequency of errors in refraction in the eyes of lower animals, a knowledge of refraction is helpful in veterinary medicine when it is desirable to examine the optic fundus. Visualization of the fundus is an aid in differential diagnosis of certain nervous diseases. It is also the only available means of definitely detecting lesions in this portion of the eye during life.

Ophthalmoscopic Examination

Examination of the optic fundi of lower animals by the aid of an ophthalmoscope usually is not difficult when the correct methods are employed. The examination should be made in a darkened room or stall. This will eliminate objectionable shadows and reflections from other sources of light and usually will also cause sufficient physiologic dilatation of the pupil to permit satisfactory examination of the fundus without the use of mydriatics. If it is necessary to use atropine or some other mydriatic, the pupillary reflexes should be noted before the drug is used. In the presence of certain nervous diseases, or if the iris is diseased, edematous or adherent to the lens capsule, dilatation of the pupil may be impossible. Examination of the optic fundus may then be impossible or unsatisfactory.

Proper restraint of the subject is, of course, essential for safety and convenience. Small animals should be placed on a table or a box. I frequently administer a general anesthetic to nervous or irritable dogs.

The ophthalmoscope should be held close to the eye of the subject about 4 cm. from it and the eye of the examiner must be held close to the lens aperture of the instrument. I always begin observations through the flat (o) lens of the ophthalmoscope. If the retina and optic papilla can not be properly visualized through this lens, I rotate the lens holder of the instrument and try the various biconcave (—) and biconvex (+) lenses and select for immediate use the one that gives the best results. The lens and inner structure of each eye should be carefully examined. The lens and vitreous body should be clear and colorless. If bodies are present, note if they are mobile or stationary. The former occur in the aqueous humor and vitreous body, while the latter occur in the cornea and lens. The retina, arteries, veins and optic disk should be carefully observed. The choroid should be visible through the retina, causing the latter to appear dark. Hemorrhages usually are serious. Light regions in the retina may be attributable to detachment or other destructive disease of the retina or choroid. Subretinal exudation is most frequently observed in horses. It commonly causes bulging of the retina. The optic papilla should be sharply outlined and slightly depressed. If it is bulging and the margin is irregular or indistinct, the condition is termed a choked disk. This commonly is caused by increased intracranial pressure from any cause. It also may be associated with encephalitis.

If glaucoma is present, the optic papilla may be markedly depressed. The presence or absence of glaucoma usually can be determined by testing the tonicity of the eyeball. This can be determined by placing the finger tips upon the upper eyelids and pushing the eyeball down upon the floor of the orbit.

The vessels in the fundus can be easily visualized by the aid of an ophthalmoscope. They should appear smooth and free from constrictions and be quite straight as they emerge from the papilla.

Naked-Eye Examination

The naked-eye method of examination of the eyes and eyelids, if properly employed, will reveal all gross lesions that are situated anterior to and, in some instances, in

the lens. To facilitate this examination, the subject should be placed in a doorway or in front of a window facing a single source of light. Carefully note the eyelids, eyelashes, conjunctiva, character of the lacrimal secretions and the pupillary reflexes. Examine the cornea, iris, lens and aqueous humor by both direct and transverse illumination. Scars on the posterior surface of the cornea usually are more visible by transverse than by direct illumination. The aqueous humor should be examined for the presence of blood, pus or inflammatory exudate.

An increase in the angle of the upper eyelid frequently is a manifestation of disease such as impaired vision from any cause or atrophy of the eyeball, muscles or orbital fat. In the horse it is a common symptom of previous disease of the eye. The eyelids should be examined for the presence of defects such an entropion, ectropion, scars, cysts and neoplasms. When examining the eyelids of a dog, one must remember that drooping of the lower eyelids is normal and desirable in bloodhounds and Saint Bernards, but it is an undesirable defect in the other breeds. Entropion occurs most frequently in chows and is a common cause of traumatic conjunctivitis in this breed.

The conjunctiva should be examined for the presence of the inflammatory lesions, foreign bodies, dermoid and other cysts and various neoplasms.

CONJUNCTIVITIS

Conjunctivitis without doubt is the most frequent disease of the eyes observed in lower animals. It is manifested by congestion and exudation. It may be acute, subacute or chronic and simple or purulent. Conjunctivitis commonly occurs secondarily with certain general infectious diseases. In the absence of some obvious systemic disease one should look for a local cause for the inflammation. But simple conjunctivitis can result from exposure to strong light or wind alone. It is common in breeds of dogs having protruding or bulging eyes. It is also frequent in dogs that are carried in automobiles, especially those that sit with the head out of an open window.

When treating conjunctivitis, one should always seek the cause and remove it if possible. For direct application, I prefer oily preparations such as mineral oil and the various ophthalmic ointments. Plain, unmedicated mineral oil is preferable to water for removing secretions from the eye and in cases of simple conjunctivitis this usually is sufficient to cause prompt relief. Aqueous solutions too frequently irritate more than is desirable. However, some animals do very nicely following treatment with aqueous solutions.

THE CORNEA

The cornea is a transparent fibrous membrane composed of five histologic layers: the anterior epithelium, anterior elastic membranes, substantia propria, posterior elastic membrane and the posterior endothelium. It contains no blood vessels, as they would interfere with light transmission. The cornea closes the anterior portion of the eyeball and thus serves as both a window and a protective membrane.

Various lesions of the cornea are described in the literature. Because of its exposed position, traumatic lesions are frequent. But, because of the transparent character of the cornea and the absence of blood vessels and hemorrhage, small abrasions are difficult to visualize by ordinary methods. They can, however, be readily stained and visualized by the application of an aqueous solution of fluorescein. I use a 2 per cent solution. One drop of this is placed on the cornea and the surplus is immediately washed off with sterile saline solution. If an abrasion or a scar is present, it will appear green.

Inflammatory disease of the cornea is termed keratitis. The causes of keratitis are various. The lesion may be localized or diffuse and superficial or interstitial. Superficial lesions frequently result in ulceration of the cornea. This combination is commonly termed ulcerative keratitis. This disease is more frequent in dogs and cattle than in other lower animals. It may occur as a primary contagious disease in

cattle. A condition simulating interstitial keratitis can be produced by merely increasing the intraocular pressure in a normal eye. If the increase in the intraocular pressure is not too severe, the capacity will disappear instantaneously when the pressure is released. It is possible that some cases of bilateral keratitis are due to spontaneous transient glaucoma.

Various methods of treatment of keratitis have been suggested. All apparently have some merit, but none are specific in all cases. Foreign protein therapy is perhaps most frequently employed. When the lesion in the cornea is associated with uveitis, this treatment appears to be of definite value. Various specific ointments and pastes have been used with occasional excellent results. Wittmann and Wohgeld[1] described iontophoresis in the treatment of certain lesions of the cornea. This treatment consists in applying an electrode to the anesthetized cornea for the purpose of bringing the ions in the remedy into action in the diseased tissue. Reihart,[2] in 1933, reported a new method of treating both corneal opacities and cataracts. This treatment consisted in subconjunctival injections of a product known as Phenolaine. Good results were reported. Labedz[3] obtained excellent results by treating interstitial keratitis with small doses of roentgen rays of medium penetration administered at intervals of two to three weeks. He reported eight cases in which this treatment was successful. Reihart,[4] in 1932, described a method of surgical treatment of chronic ulcerative keratitis. This consisted in placing a strip of flap of conjunctiva over the crater of the ulcer. Cauterization of ulcerative lesions on the cornea with caustic chemicals or thermal units has given good results in the hands of some operators. But I believe one should not expect a single method of treatment to be specific for all cases. I usually begin by using foreign protein therapy and the local application of ointment.

Protective dressings and shields or collars to prevent accidental injury or self-mutilation are advisable in some cases. I have learned that dogs are less apt to injure an affected eye if anesthetic ointments are avoided.

If rupture of the cornea occurs and the iris prolapses into or through the wound, healing is retarded. The protruding or adherent portion of the iris should be resected. If the eyeball remains relatively free of infection, the ruptured cornea will heal and result in a good cosmetic effect, even though sight is impaired or destroyed. When the eyeball is badly infected, surgical removal of the eye usually is advisable.

Uveitis

Inflammation of the vascular membranes of the eye (iris, ciliary body and choroid) is termed uveitis. This occurs frequently in some breeds of animals. So-called recurrent opthalmia of horses is a form of uveitis. It has been referred to as anterior uveitis. But since inflammation of the iris, lens capsule and lens is common in recurrent ophthalmia, the term iridocyclitis is more specific. Cataract is a frequent permanent residual lesion of uveitis and iridocyclitis. Uveitis is always a serious disease because it can also cause various other permanent lesions. Since the posterior surface of the iris is normally in contact with the anterior surface of the lens, adhesions (posterior synechia) are common. These may break later, leaving small deposits of the iris on the lens capsule as evidence of previous disease. Detachment of the retina due to subretinal exudation or hemorrhage is not infrequently observed in severe cases of uveitis. This commonly causes atrophy of the detached portion of retina.

The causes of uveitis undoubtedly are multiple. It is my opinion that infectious uveitis is most frequent and I usually treat all cases as if they were known to be infectious. There is no one specific treatment for all cases of uveitis, but this is one disease in which foreign protein therapy seems to be indicated. It usually is beneficial and in many cases is specific. I com-

monly use sterile milk. This is injected intramuscularly. A dog would receive 1 to 5 cc., depending upon size and age. I use 3 to 10 cc. in horses. Injections are repeated in two to three days if necessary. Colonel Underwood, of the United States Army, informed me recently that he has used typhoid vaccine intravenously in the treatment of recurrent ophthalmia in horses and has obtained excellent results. Since then, I have used it in a small series of cases and likewise have obtained good results. I have injected as much as 3 cc. intravenously in one dose. The correct dose, however, has not been determined. I always have the subject placed in a darkened stall or cage and apply atropine ointment to the affected eye as indicated. To avoid synechia, one should permit the pupil to contract before a second application of this ointment is made. The oral administration of sodium iodide or potassium iodide appears to be beneficial in some cases.

Various other foreign proteins and vaccines have been recommended and employed by other authors. All appear to have merits.

CATARACT

Cataracts are lesions in the lens capsule or lens. They occur most frequently in horses and dogs. In horses they usually result from inflammatory disease of the eye, uveitis or iridocyclitis. In dogs they are commonly a manifestation of senility.

The treatment of cataract in lower animals is generally unsuccessful. I found detachment of the retina in association with cataract in nearly all of the affected eyes of horses I examined. It is obvious that removal of the cataract would not restore sight in these cases. Since absorption of the lens apparently does not occur in dogs, discussion would be useless in this species of animal. Surgical removal of the lens or causing it to prolapse into the fundus of the eye appears to be the only treatment of cataract that might be beneficial in the dog.

Primary inflammation of the sclera, scleritis, probably does not occur in lower animals. At least, I have found no actual case reports of this disease.

CONGENITAL DEFECTS

Various congenital defects are known to occur in lower animals. Undoubtedly ectropion, entropion and dermoids are the most frequent and important defects observed. Because of the frequency of prolapse of the glands of Harder in some breeds of dogs, it, too, might be classed as a congenital defect. All of these defects can be treated or corrected surgically. Dermoids and prolapsed lacrimal glands are excised. In excising the latter, one should carefully dissect them free from the cartilage of the membrana nictitans. If too much tissue is removed, the eyeball may appear atrophic and the lower eyelid may droop.

Ectropion can be satisfactorily corrected by excising a small section of skin at the lateral canthus. The site of the excision and the quantity of skin necessary to excise can be determined through trial and error by applying a forceps on a fold of skin at the proposed site of operation.

Entropion usually can be corrected by excising a section of skin from the affected eyelid, causing eversion of the lid margin.

Many other diseases of the eyes and their appendages have been observed and described in the literature. They, too, are important but many are of infrequent occurrence. I shall not attempt to discuss them at this time.

REFERENCES

[1]Wittmann, and Wohlgeld, H.: Iontophoresis in the treatment of diseases of the cornea. Ztschr. f. Veterinark., xlvi (1934), pp. 305-321.

[2]Reihart, O. F.: A new method of treating corneal opacities and cataract. North Amer. Vet., xiv (Oct., 1933), pp. 47-48.

[3]Labedz, M.: Radio therapy in interstitial keratitis of dogs. Abst., North Amer. Vet., xiv (Jan., 1934), p. 57.

[4]Reihart, O. F.: Conjunctival keratoplasty. North Amer. Vet., xiii (Jan., 1932), pp. 57-58.

But for the control of animal plagues through the police measures now in force, there would not be enough food to nourish the present population of the world.

Sulfured Soil for Poultry Yards*

By M. W. EMMEL, *Gainesville, Fla.*
Florida Agricultural Experiment Station

In various sanitary programs, houses, drinking and feeding utensils can be disinfected, but there has been no practical means of disinfecting soil. This has been a serious difficulty for many poultrymen who find it necessary to confine their birds in one yard all of the time. Poultrymen who have the advantage of large acreages find it profitable, from a disease-control standpoint, to move colony houses to new areas or to rotate their flocks through a series of yards at regular intervals. This is impossible in the case of the poultryman who does not have such acreages available.

During the past three years, experiments have been conducted at this station on various uses of sulfur in connection with poultry raising. It has been found that sulfured soil offers possibilities of being of practical assistance in disease control on premises in which birds must be confined to the same yards continuously.

Commercial flour sulfur has been used for this purpose. This type of sulfur is ground, run-of-mine sulfur grading 90 per cent through 80-mesh screen. Commercial flowers of sulfur is a refined product selling at about twice the price of commercial flour sulfur. Except for its higher cost, flowers of sulfur is suitable also for treating soil.

Commercial flour sulfur is broadcast over the surface of the soil at the rate of 800 pounds per acre (1.9 pounds of sulfur per 100 square feet) and mixed with the top soil with a rake. The action of soil bacteria on sulfur produces sulfuric acid, which renders the soil acid in reaction. In experimental tests approximately two to two and one-half months are required for the maximum acidity of a pH of 2.0 to 2.2 to be reached in the surface soil (0-3 inches). Increased acidity developed more rapidly during the summer months than during the winter months. In certain soils the pH was readily reduced to 3.5 but considerable time was required for increased acidity to develop. This is apparently due to the necessity of disrupting the buffer system of the soil. Eddins[1] has reported that the application of 800 pounds of commercial flour sulfur per acre reduced the pH from 1.3 to 1.7 points on three different types of sandy soils, the maximum acidity being a pH of 3.5. The sulfur, however, was mixed with the soil to a depth of six inches. In the experiments being reported, sulfur was mixed with the surface soil, which resulted in a greater concentration of sulfur in the surface soil than in the experiments of Eddins.

Sulfured soils treated as described have remained at the maximum acidity for one year and free sulfur could still be found in the soil. Thus, but one application a year has been necessary. As a general rule, after rains, there is a slight raise in pH value of the soil which possibly is due to leaching of acid from the soil. However, shortly thereafter the pH value returns to its original status.

The application of sulfur did not change the character of the sandy soils used in these experiments. However, it should not be overlooked that such treatment may change the character of other types of soils. This is true particularly of highly buffered soils which are common in many other states. On highly buffered soils, it also may be necessary to repeat the above dosage of sulfur to disrupt the buffer system to bring the pH to the desired level.

Acidity of sulfured soil is so increased that practically all forage will cease to grow. Thus soil which harbors desirable trees or shrubs should not be treated. In case it is desirable to place sulfur-treated soils in cultivation, the application of lime will neutralize the acidity. Either hydrated

*Received for publication, December 12, 1938.

lime or agricultural limestone may be used for this purpose. When the lime is finely divided and the soil is moist, neutralization occurs rapidly. For practical purposes the lime should be incorporated thoroughly into the soil several weeks prior to planting. Three times as much ground limestone or one and one-half times as much hydrated lime should be added as sulfur originally applied to the soil.

Experimentally and in the field, the sulfured soil has been found to be of practical assistance in the control of intestinal parasites. The increased acidity results in the disappearance of all of the intermediate hosts of poultry tapeworms (snails, slugs, earthworms, etc.), whose life cycle definitely is associated with the soil. The life cycle of the common roundworm, whose egg must undergo incubation over a period of time on the ground, apparently is retarded. Pathogenic bacteria probably do not remain viable as long as the pH values of sulfured soil as in cases in which the soil is not so treated. Eddins[2] has reported the control of brown rot (a bacterial disease of Irish potatoes) by adding sulfur to soil immediately after the potato crop is harvested. The microörganism will not remain viable below pH 4.0. The land was utilized the following year for potatoes without the appearance of brown rot after the application of the proper amount of lime to the soil.

Since the above procedure has proven of practical value to poultrymen whose birds are confined to single yards all of the time, it appears to the author that this procedure may be of value on fox farms. On most fox farms and on some poultry farms wire netting extends from fences into the soil. Since soil acidified with sulfur will cause buried wire netting to rust rapidly, it is advisable to use some other barrier in soils treated with sulfur.

The author wishes to emphasize that while the sulfurization does not absolutely disinfect soil, it offers a more practical and economical method of soil sanitation than other methods now in use.

REFERENCES

[1]Eddins, A. H.: Adjusting pH reactions of soils with sulfur and limestone to control brown rot of potatoes.

[2]Eddins, A. H.: Brown rot of Irish potatoes and its control. Fla. Agr. Exp. Sta. Bul. 299 (1936).

Perosis a Manganese Deficiency

That perosis (slip tendon) is a manganese deficiency seems to be a justifiable conclusion. The manganese-deficiency theory of this common affliction of chickens has been confirmed at a number of different experiment stations: New York, Oklahoma, Michigan, New Hampshire, *et al.* The amount of manganese (chloride) required to prevent perosis is 50 parts to the million. This element is necessary for growth, egg production and hatchability (Card, 1938).

Fewer hens and more eggs per hen is given as one of the causes of the low prices of eggs this winter and spring.

Let geese incubate their own eggs and brood their own goslins.—*The Poultry Tribune.*

The World's Poultry Congress is being financed entirely by the poultry industry. The $100,000 appropriated by Congress can be used only for the government's participation. The income of the Congress is derived from the sale of exhibit space and memberships.

The rate of depletion of riboflavin (vitamin G) in hens is rapid and the loss causes a decrease in hatchability. The reserve can, however, be quickly built up by supplementing the ration with synthetic riboflavin or protein-free riboflavin extract. —*L. E. Card, in the Poultry Tribune.*

Cod liver oil and other fish oils have become almost universal articles of diet in fowl because they are a practical source of vitamins A and D. In time, says a well know authority, wheat germ oil will also be a universal ingredient of the poultry ration.

Chemical Changes in the Blood of Swine Infected with Hog Cholera*

By D. F. EVELETH *and* L. H. SCHWARTE, *Ames, Iowa*
Veterinary Research Institute, Iowa State College

Studies of the chemical changes of the blood of cholera-infected swine are not numerous. Roderick and Schalk[1] made a few blood analyses for nonprotein nitrogen, urea and sugar of normal and cholera-infected swine. Their data show little difference in the blood sugar of the two groups. There was evidence of nitrogen retention in some of the cholera-infected swine as shown by high urea and non-protein nitrogen values. Shope[2] determined cholesterol and cholesterol esters of the plasma of swine artificially infected with cholera virus. By repeated examinations of plasma from the same animals during the course of the disease, he found a preliminary hypocholesterolemia during the incubation period, followed by a hypercholesterolemia after the onset of clinical symptoms. Later, in his surviving animals, another period of hypocholesterolemia developed. Hewitt[3] compared some of the whole-blood, nonprotein nitrogen constituents and sugar as well as the plasma inorganic phosphorus and calcium of normal and cholera-infected swine.

Quoting from Hewitt's comparison of average values (table 41, p. 203):

Constituents	Mg. Per Cent	
	Normal Swine	Cholera Swine
Total N. P. N. whole blood	31.89	37.02
Urea N. whole blood	7.42	3.84
Uric acid whole blood	1.94	1.92
Creatinine whole blood	1.69	2.21
Sugar whole blood	125.95	71.38
Inorganic plasma	7.22	6.4
Calcium plasma	12.39	14.01

These data indicate a slight nitrogen retention but with a definitely decreased urea nitrogen, an increase in creatinine, a reduction in sugar and phosphate and an increase in calcium. Hewitt ascribes the decrease in urea and sugar to anorexia.

Oglesby, Hewitt and Bergman[4] found that the volume of cells was 47.08 per cent for normal swine blood and 37.38 per cent for blood of cholera-infected animals.

Zuverkalor and Kucherendo[5] report an increase in the serum proteins of hyperimmunized swine with a preliminary fall in the globulin followed by a definite rise.

Materials and Methods

As a part of investigations under way in this laboratory, blood studies have been made on certain experimental cases of cholera. These data are presented for individuals rather than averages. The methods used for analyses are listed as follows:

Hematocrit[6] Wintrobe tubes were used and sample centrifuged to constant cell volume. Phosphorus, the method of Fiske and Subbarow[7] was used in certain cases but the majority of the samples were analyzed by a modification developed in this laboratory in which the organic matter was ashed with nitric and perchloric acids[8] and the phosphorus determined on the dry residue with the reagents of Fiske and Subbarow.

N. P. N.	Folin Wu method[9]
Urea N.	Eveleth[10]
Amino Acid N.	Folin[11]
Uric Acid	Benedict[12] by direct color development
Creatinine	Folin Wu[9]
Cholesterol	Myers and Wordell[13]
Albumin, globulin and total proteins	Greenberg[14]
Sugar	Benedict[15]
Chlorides	VanSlyke[16]
CO_2	VanSlyke and Neill[17]
Fibrin	Direct precipitation by $CaCl_2$ and micro Kjeldahl

*Received for publication, September 29, 1937.

Calcium	$KMnO_4$ titration after precipitation from a trichloracetic acid filtrate
Magnesium	Precipitation as $MgNH_4PO_4$ and colorimetric estimation of the phosphate[7]

It has been the policy in all blood studies to fast swine for at least 14 hours before obtaining blood samples. This procedure allows complete digestion and absorption of nutrients and establishes a uniform set of conditions.

The method of obtaining blood has varied with the future use to be made of the animal. Tail-bleeding was employed unless the animals were to be killed, in which case a bleeding cannula was introduced directly into the heart. In all cases every effort was made to keep the animals from struggling.

Sodium citrate was used as an anticoagulant, 300 mg. of the crystals to 100 cc. of blood. With proper mixing, no trouble from clotting was encountered.

All animals were kept in isolated units for at least a week before being inoculated intramuscularly with hog-cholera virus. Temperatures were taken daily, symptoms observed, and all animals were examined postmortem. The cases reported here are considered as hog cholera, uncomplicated by other pathologic conditions, based upon postmortem examination.

In order to get some conception of changes in the serum of cholera-infected swine, the following experiment was conducted. Animals 4009 and 4010 were kept indoors for a period of several weeks. Animal 4009 was used in a cholera experiment and found to be immune. Later, the same animal was used to furnish blood in some experiments. After a lapse of several weeks, in which both 4009 and 4010 were kept under identical conditions, 4010 was injected with cholera virus. Seven days after injection, both animals were killed and a direct comparison of certain blood-serum constituents made. These data are presented in table I and indicate certain changes that might be ascribed to the disease. Analyses were made of the blood and serum or plasma of other cholera-infected animals killed from time to time. In comparing these data (table II), with those obtained on normal animals, certain trends in the variation of different constituents were noted, but the variations within the group are extremely wide.

TABLE I—*Comparison of serum of normal and cholera injected pigs.*

SWINE NO.	4009 NORMAL	4010 CHOLERA
CONSTITUENTS	MG. PER CENT	
Serum inorganic p.	6.66	5.92
Urea n.	9.00	12.80
Uric acid	1.79	1.54
Amino acid n.	10.60	5.18
N. P. N.	30.90	34.40
Creatinine	1.39	1.61
Cholesterol	143.	177.
Lipid p.	7.00	4.00
	PER CENT	
Serum albumin	3.32	3.04
Globulin	3.48	3.12
Total protein	6.80	6.16

The data of Shope suggested approaching the chemical pathology by periodic bleedings of the same animal during the course of the disease. This procedure was followed on animals 6133, 6134, 6135 and 6136. These analyses are given in tables III, IV, V and VI. Data for the cell concentration of a given constituent have been calculated by means of the following formula:

$$C(\text{conc.}) = \frac{W\,B\ \text{conc.} - (P\ \text{conc.} \times 1 - H}{H}$$

C = cells W B = whole blood P = plasma

H is expressed as the decimal fraction and the concentration of the constituent is expressed as mg. per cent. The total phosphorus data have been obtained by addition of the A. S. P. and lipid fractions. In most cases the value so obtained has agreed very closely with actual determination of the total P.

A second series of determinations were made on swine 6143 and 6144. These animals were much larger than the animals

TABLE II—*Analyses of blood of swine infected with cholera.*

SWINE	4708	4711	4714	6086	6087	6094
Survival time—days	8	11	12	10	8	11
Whole blood						
Hematocrit	44.9		43.3	46.0	46.7	
N. P. N.—mg. per cent	28.6	40.0	50.0			
Urea n.—mg. per cent	5.5		26.0			
Amino acid n.—mg. per cent	6.7	6.2	9.3			
Uric acid—mg. per cent	1.6	2.2				
Creatinine—mg. per cent		1.6	1.5			
Inorganic p.—mg. per cent	4.6	4.1	3.9	7.2	9.2	8.9
Lipid p.—mg. per cent	11.2	7.6	6.5	8.8		6.2
A. S. P.—mg. per cent		50.0				29.0
Sugar—mg. per cent	102.0	45.0				
Cholesterol—mg. per cent	150.0	105.0	174.0			
Magnesium—mg. per cent				5.4	7.0	5.0
Plasma						
Fibrin—per cent			0.72			
CO^2 Content—vol. per cent	47.5					
CO^2 Capacity—vol. per cent	51.3					
Serum						
Total protein—per cent	5.4		7.1	7.4		3.8
Albumin—per cent	3.3		3.8			2.9
Globulin—per cent	2.0		3.3			.9
Inorganic p.—mg. per cent	5.6			6.5	8.6	5.8
Lipid p.—mg. per cent				5.1		3.9
A. S. P.—mg. per cent		10.1				5.8
Calcium—mg. per cent	9.0			12.0	11.1	
Magnesium—mg. per cent				2.4	2.2	2.7
Amino acic n.—mg. per cent			6.7			
Cholesterol—mg. per cent			167.0			

TABLE III—*Blood changes in cholera swine—6133.*

Date	8/15	8/18	8/21	8/24	8/26*
Temperature	102.2	106.2	107.4	107.2	107.0
Days after injection	0	3	6	9	11
Hematocrit	42.5	40.7	32.8	37.4	33.7
	MG. PER CENT				
Constituent					
Whole blood total p	50.9	48.9	39.0	41.2	46.6
Plasma total p	12.2	10.4	8.5	9.7	9.3
Cell total p	103.	105.	101.	94.	120.
Whole blood a. s. p.	42.0	42.2	32.0	32.0	38.3
Plasma a. s. p.	6.9	8.4	5.2	5.1	5.3
Cell a. s. p.	89.6	91.4	84.0	76.9	103.2
Whole blood lipid p	8.9	6.7	7.0	9.2	8.3
Plasma lipid p	5.3	2.0	3.3	4.6	4.0
Cell lipid p	13.8	11.0	13.1	16.8	16.9
Whole blood amino acid n	9.0	10.0	7.6	7.3	9.0
Plasma amino acid n	6.4	8.0	6.4	8.2	7.3
Cell amino acid n	12.5	13.0	14.6	5.9	12.5
Whole blood magnesium	6.6	6.2	6.1	6.3	6.7
Plasma magnesium	3.3	3.4	3.1	3.1	3.6
Cell magnesium	11.0	10.3	12.2	11.8	12.7
Whole blood chlorides	268.	254.		256.	266.
Plasma calcium	9.9	10.3	7.8	5.4	6.2

*Animal destroyed.

TABLE IV—*Blood changes in cholera swine—6134.*

Date	8/15	8/18	8/21	8/24	8/26*
Temperature	102.4	106.4	107.2	107.2	107.0
Days after injection	0	3	6	9	11
Hematocrit	42.5	43.8	37.1	37.0	35.7
Constituent	MG. PER CENT				
Whole blood total p	50.8	47.8	47.0	40.7	46.8
Plasma total p	11.2	9.5	10.7	9.6	11.7
Cell total p	104.	97.1	109.0	94.0	110.
Whole blood a. s. p.	41.3	42.2	39.1	30.5	38.7
Plasma a. s. p.	5.9	7.3	7.4	5.3	6.6
Cell a. s. p.	89.2	87.0	93.0	73.6	96.8
Whole blood lipid p	9.5	5.6	7.9	10.2	8.1
Plasma lipid p	5.3	2.2	3.3	4.3	5.1
Cell lipid p	15.0	10.1	14.8	20.3	13.4
Whole blood amino acid n		8.8	7.8	10.4	9.3
Plasma amino acid n	6.1	8.5	6.7	7.2	9.3
Cell amino acid n		8.9	7.5	15.9	9.3
Whole blood magnesium	6.6	7.0	7.3	6.7	6.7
Plasma magnesium	3.4	3.1	3.3	3.4	3.4
Cell magnesium	10.8	12.1	13.2	12.4	12.6
Whole blood chloride	261.	266.		262.	252.
Plasma calcium	9.9	9.9	7.7	6.4	8.2

*Animal destroyed.

TABLE V—*Blood changes in cholera swine—6135.*

Date	8/15	8/18	8/21	8/24	8/26*
Temperature	102.3	105.8	107.6	107.4	107.0
Days after injection	0	3	6	9	11
Hematocrit	42.2	39.0	36.5	35.6	35.3
Constituent	MG. PER CENT				
Whole blood total p	50.7	44.3	46.9	38.8	45.4
Plasma total p	11.8	9.3	9.6	9.5	9.5
Cell total p	104.0	98.9	112.	92.	111.
Whole blood a. s. p.	41.8	37.6	39.1	31.4	36.7
Plasma a. s. p.	6.1	7.0	6.0	5.3	5.5
Cell a. s. p.	90.7	85.3	96.5	78.6	93.8
Whole blood lipid p	8.9	6.7	7.8	7.4	8.7
Plasma lipid p	5.7	2.3	3.6	4.1	4.0
Cell lipid p	13.2	13.6	15.1	13.5	17.3
Whole blood amino acid n	8.8	10.3	8.0	8.4	7.4
Plasma amino acid n	6.0		6.1	7.8	6.5
Cell amino acid n	12.5		11.2	9.5	8.7
Whole blood magnesium	7.0	6.7		7.3	6.8
Plasma magnesium	3.4	3.2	3.2	3.5	3.5
Cell magnesium	12.1	12.3		14.3	12.7
Whole blood chloride	254.			265.	261.
Plasma calcium	9.4	8.9	7.8	6.5	7.8

*Animal destroyed.

TABLE VI—*Blood changes in cholera swine—6136.*

Date	8/15	8/18	8/21	8/24	8/26*
Temperature	102.8	105.2	107.2	107.0	107.0
Days after injection	0	3	6	9	11
Hematocrit	41.8	39.3	37.5	35.6	33.3
	Mg. Per Cent				
Constituent					
Whole blood total p	55.4	44.4	49.8	40.3	43.3
Plasma total p	11.5	9.0	8.6	8.7	8.3
Cell total p	117.	99.	118.	97.	114.
Whole blood a. s. p.	46.5	37.2	41.7	32.6	37.3
Plasma a. s. p.	5.8	6.8	5.2	4.7	6.0
Cell a. s. p.	104.	84.2	102.9	83.2	100.
Whole blood lipid p	8.6	7.2	8.1	7.7	6.0
Plasma lipid p	5.7	2.2	3.4	4.0	2.3
Cell lipid p	12.7	15.0	16.0	14.3	13.5
Whole blood amino acid n	9.0	9.4	7.6	7.8	8.6
Plasma amino acid n	6.1	7.4	6.7	6.8	7.2
Cell amino acid n	13.1	12.4	9.1	9.5	11.4
Whole blood magnesium	7.5	7.1	6.9	7.2	6.6
Plasma magnesium	3.3	2.9	3.4	3.4	
Cell magnesium	13.4	13.5	12.8	14.0	
Whole blood chloride		264.		254.	238.
Plasma calcium	6.8	11.4	8.4	6.2	

*Animal destroyed.

TABLE VII—*Comparative variations in the blood constituents as the result of fasting swine and infection with hog cholera swine—6143.**

Date	9/17	9/24	9/29	10/5
Temperature	101.4	101.4	105.4	107.
Days of fasting	0	7		
Days after virus inoculation		0	5	11
Hematocrit	43.2	44.5	34.0	27.2
	Mg. Per Cent			
Constituent				
Whole blood total p	38.4	44.2	38.4	37.0
Plasma total p	10.4	10.7	9.6	11.8
Cell total p	75.0	86.0	94.0	104.
Whole blood a. s. p.	31.6	35.9	32.0	31.5
Plasma a. s. p.	6.7	6.4	7.0	6.0
Cell a. s. p.	63.9	72.4	80.6	99.5
Whole blood lipid p	6.8	8.3	6.4	5.5
Plasma lipid p	3.7	4.3	2.6	5.8
Cell lipid p	10.8	13.2	13.8	4.8
Whole blood inorganic p	6.0	8.4		
Plasma inorganic p	6.1	6.2		
Cell inorganic p	5.8	11.2		
Whole blood amino acid n	10.4	8.3	9.3	9.1
Plasma amino acid n	7.5	8.0	8.0	8.9
Cell amino acid n	14.1	8.8	13.2	9.5
Whole blood magnesium	4.9	4.5	3.8	3.5
Plasma magnesium	2.4	2.3	2.5	2.2
Cell magnesium	8.3	7.2	6.5	7.0
Whole blood chloride	258.0	269.0	296.0	264.0
Plasma calcium	10.7	10.8	8.3	8.5

*Animal died, October 7, 1936.

TABLE VIII—*Comparative variations in the blood constituents as the result of fasting swine and infection with hog cholera swine—614.*

Date	9/17	9/24	9/29	10/5	10/8*
Temperature	101.2	101.2	106.0	107.2	106.6
Days of fasting	0	7			
Days after virus inoculation			5	11	14
Hematocrit	41.6	45.0	40.2	35.0	28.
	Mg. Per Cent				
Constituent					
Whole blood total p	38.9	38.0	38.3	39.0	41.6
Plasma total p	11.1	11.0	10.2	11.0	12.7
Cell total p	80.0	71.0	80.0	91.0	116
Whole blood a. s. p.	31.6	30.3	31.4	32.4	35.9
Plasma a. s. p.	7.5	6.9	7.4	6.4	7.4
Cell a. s. p.	65.3	58.8	67.2	80.6	109.4
Whole blood lipid p	7.3	7.7	6.9	6.6	5.7
Plasma lipid p	3.6	4.1	2.8	5.0	5.3
Cell lipid p	12.5	12.0	12.9	9.7	6.8
Whole blood inorganic p	6.3	8.3			
Plasma inorganic p	6.7	6.4			
Cell inorganic p	5.8	10.7			
Whole blood amino acid n	10.2	9.1	9.3	9.4	8.9
Plasma amino acid n	8.0	7.9	7.9	8.1	
Cell amino acid n	13.2	10.7	11.5	11.1	
Whole blood magnesium	5.0	4.1	4.1	4.0	5.4
Plasma magnesium	2.4	2.4	2.8	2.3	2.7
Cell magnesium	8.0	8.4	8.4	10.0	12.5
Whole blood chloride	268.	281.	275.	269.	274.
Plasma calcium	11.3	10.5	7.9	8.9	6.0

*Animal destroyed.

in the previous group and the initial values for certain constituents are different. A preliminary sample was obtained and the pigs were fasted seven days in order to determine the effect of starvation. Water was allowed *ad libitum*. On the seventh day, the animals were injected with a virulent virus and allowed feed during the remainder of the experiment. Blood samples were always drawn prior to feeding. The analyses are summarized in tables VII and VIII.

Other animals have been used, but the data are very similar to those already presented.

ACKNOWLEDGMENT

The authors are indebted to Mr. T. W. Millen and Mr. H. C. Nowlin for assistance in conducting these experiments.

SUMMARY

During the course of experimental cholera in swine, there are certain changes in the blood chemistry, particularly in the phosphorus fractions and the total plasma calcium.

DISCUSSION

In these studies the animals were always kept on clean concrete floors. Accidental infestation with parasites was avoided as much as possible, since it has been established that ascaris passing through the liver of swine greatly alter the chemistry of the blood.[18]

In reporting the concentration of a given constituent for 100 cc. of cells, there appear to be certain definite advantages. Changes observed in whole blood analyses might be entirely the effect of the progressive anemia unless reported for plasma and cells.

The changes in concentration of the various blood constituents, as shown in the tables, are difficult to interpret. Responses are different in individual animals, but definite trends seem established. There is a progressive anemia, as shown by the decreasing hematocrit value. The acid soluble phosphorus fraction of the cells increases

per unit of cells, while the plasma acid-soluble fraction tends to decrease. The lipid phosphorus of the plasma shows a definite decrease during the first three days, followed by a gradual increase. Swine 6143 and 6141 were not bled until the fifth day after infection and the lipid phosphorus of the plasma was apparently increasing at this time. The lipid phosphorus of the cells has a tendency to rise and later decrease. Here animals 6143 and 6144 showed a marked decrease after the fifth day. This may have been the effect of the preliminary starvation. The changes in amino acid nitrogen, magnesium and chlorides, are too erratic for clear interpretation.

Plasma calcium has a tendency to decrease during the course of the disease. With the limited information on calcium values in infectious diseases, an explanation of this change is impossible. It is of interest to note that Rees and Hale[19] found a decrease in serum calcium and whole blood inorganic phosphorus in cattle with anaplasmosis during the period in which clinical symptoms were manifested.

These data show various trends in changes of blood constituents. The disturbance in phosphorus and calcium metabolism are of particular interest. Shope's data show definite derangement in cholesterol metabolism and the data reported here show changes in the lipid phosphorus fraction of the cells and plasma. The rôles of these constituents in immunity are not clearly understood and more data from various infectious diseases will be necessary to establish definitely the relation of pathologic conditions to blood changes.

REFERENCES

[1]Roderick, L. M., and Schalk, A. F.: Collected studies on hog cholera. N. Dak. Agr. Exp. Sta. Bul. 210 (1927).

[2]Shope, R. E.: Variations in the plasma cholesterol and cholesterol ester content in hog cholera. Jour. Exp. Med., li (1930), p. 179.

[3]Hewitt, E. A.: Certain chemical and morphological phases of the blood of normal and cholera-infected swine. I. The concentration of certain chemical constituents. Iowa State Jour. Sci., vi (1932), p. 143.

[4]Oglesby, W. T., Hewitt, E. A., and Bergman, H. D.: Certain chemical and morphological phases of the blood of normal and cholera-infected swine. II. Certain morphological phases. Iowa State Jour. Sci., vi (1932), p. 227.

[5]Zuverkalor, D., and Kucherendo, J.: The serum protein fraction in the hyperimmunization of the pig. Chem. Absts., xxix (1935), p. 7447.

[6]Wintrobe, M. M.: A simple and accurate hematocrit. Jour. Lab. & Clin. Med., xv (1929), p. 287.

[7]Fiske, C. H., and Subbarow, Y.: The colorimetric determination of phosphorus. Jour. Biol. Chem., lxvi (1925), p. 375.

[8]Bollinger, A.: On a method for rapid digestion of biological material for analysis. Austral. Jour. Exp. Biol. & Med. Sci., x (1932), p. 57.

[9]Folin, O., and Wu, H.: A system of blood analysis. Jour. Biol. Chem., xxxviii (1919), p. 81.

[10]Eveleth, D. F.: Note on the determination of blood urea by direct nesslerization of a sodium tungstate-sulphosalicylic acid filtrate. Jour. Lab. & Clin. Med., xix (1934), p. 783.

[11]Folin, O.: A new colorimetric method for the determination of amino acid nitrogen in blood. Jour. Biol. Chem., li (1922), p. 377.

[12]Benedict, S. R.: The determination of uric acid in blood. Jour. Biol. Chem., li (1922), p. 187.

[13]Myers, V. C., and Wardell, E. L.: The colorimetric estimation of cholesterol in blood with a note on the estimation of coprosterol in feces. Jour. Biol. Chem., xxxvi (1918), p. 147.

[14]Greenberg, D. M.: The colorimetric determination of the serum proteins. Jour. Biol. Chem., xxxvi (1918), p. 147.

[15]Benedict, S. R.: The determination of blood sugar II. Jour. Biol. Chem., lxxvi (1928), p. 457.

[16]Van Slyke, D. D.: The determination of chlorides in blood and tissues. Jour. Biol., lvii (1923), p. 523.

[17]Van Slyke, D. D., and Neill, J. M.: The determination of gases in blood and other solutions by vacuum extraction and manometric measurements. Jour. Biol. Chem., lxi (1924), p. 523.

[18]Biester, H. E., and Eveleth, D. F.: Blood and tissue studies in experimental ascariasis. Amer. Jour. Hyg., xxv (1937), p. 135.

[19]Rees, C. W., and Hale, M. W.: Morphological and chemical studies of the blood of cattle in health and during anaplasmosis. Jour. Agr. Res., liii (1936), p. 477.

Hep and Zep, two of the Brookfield (Ill.) Zoo's prized penguins, were forced to step out of the penguin parade recently. They frowned, limped, hobbled and squawked. The reason—corns. But now the corns have been removed and Hep and Zep strut proudly along with their colleagues.

White rats can be made dead drunk merely by smelling alcohol fumes, the University of North Carolina has announced. As far as is known, this is the first time that drunkenness has been produced scientifically by simply smelling the drink.

Anaphylaxis as Related to Biologic Prophylaxis and Treatment of Animals*

By JOHN REICHEL, *Glenolden, Pa.*

Your Secretary asked me to participate in this program and suggested that I review the subject of anaphylaxis. It was further stated that anaphylaxis is of general interest to the practitioner, especially in connection with the use of biological products in the prophylaxis and treatment of animals. This is not a new subject, and much has been recorded in the literature regarding the manifestations of anaphylaxis and attempts toward its reproduction experimentally, with a view of determining its causes, nature and effects.

Hoskins,[1] in 1919, published a comprehensive review on "Anaphylaxis in Veterinary Practice." In this review the following statements are worth repeating, inasmuch as they still hold true:

> Anaphylaxis is a term coined by a French investigator, Charles Richet, whose investigations as far back as 1902 really laid the foundation for our present knowledge of the subject. * * * *
>
> Today the word really has a double meaning, (a) the phenomena or symptoms of intoxication exhibited by a sensitized animal following the second injection of a foreign protein after an appropriate interval, or (b) the state of being sensitized to a foreign protein.
>
> In bringing about an anaphylactic state, there are three distinct steps: (1) sensitization; (2) incubation; and (3) intoxication. Proteins, of which there is an endless variety, seem to be the only substances that are capable of bringing about the anaphylactic state, and these proteins must be foreign to the animal receiving them, or anaphylaxis will not result. * * * *
>
> All species of animals appear to be capable of sensitization. Most observations have been made on the guinea pig, rabbit and dog, although the literature on the subject contains references to other animals, including the horse, cow, goat, sheep, pig, rat, white mouse, frog and various birds. Of the laboratory animals, the guinea pig is probably the best for demonstration purposes, and the injections of the protein should be made subcutaneously, intravenously or intraperitoneally.
>
> The incubation period in anaphylaxis is quite similar to the incubation period of an infectious disease. The period varies in different species, the method or site of injections, and the quantities of the substance used. In anaphylaxis the period of incubation may be said to be the interval between the first injection of the protein material and the earliest appearance of anaphylactic shock following the second injection of the same protein material. The period varies from one week to four.
>
> * * * *
>
> That form of anaphylaxis known as "serum disease" is a phenomenon that has undoubtedly been of more concern to the practitioner of human medicine than to the veterinarian. However, in view of the fact that the underlying principles of the phenomenon are identically the same, in both man and lower animals, a brief review of the subject might be apropos at this time. The term "serum disease" has been given to anaphylaxis, because the first experiences with anaphylactic shock were had in connection with the administration of antidiphtheric serum, but before the exact nature of the trouble or the conditions surrounding it were known. As the veterinarian's experiences with anaphylaxis are quite likely to be in connection with the administration of serum, we will consider the phenomenon from this standpoint.
>
> * * * *
>
> Anaphylactic reactions are most typical when they follow the parenteral introduction of protein substances. By parenteral we mean the introduction by some route other than the digestive tract, for instance, subcutaneously, intravenously, or intramuscularly. That the substances which will produce anaphylaxis, under certain conditions, are in themselves harmless, has been proven many times. On the other hand, it is the condition of the animal or the person, with reference to the protein, which results in anaphylactic shock when the protein is injected.

Reichel and Harkins[2] published on "Peptotoxin by the Bacillus of Contagious Abortion of Cattle," in which it was shown that cattle would tolerate an initial dose of *Br. abortus* peptotoxin, but the injection of a second dose intravenously, four or five days later, would result in typical manifestations of anaphylaxis. The symptoms of anaphylaxis recorded at the time were as follows:

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

Within 20 seconds the heifer went down, showing the following reaction, which may be considered typical: Slight quivering of the muscles of the hind quarters, shoulders, or other parts of the body, lasting but a few seconds; sudden collapse and prostration; the head thrown back in an apparent attempt to straighten out the air-passages, mouth open, tongue relaxed, and hanging out, and moving back and forth; the legs extended rigidly but flexible; reflexes normal; mucous membrane noticeably paler; feces and urine likely to be voided in small amounts; pulse noticeably weakened and for a time imperceptible over the inferior maxilla, rate however not greatly affected. The reaction is most striking in the change in the respiration and this may be the only change observable in a slight reaction. In a pronounced reaction the respiratory movements are modified with the collapse. A slight slowing up may be observed before the rapid increase in a few minutes, from the normal rate of 18 to 48 to 90 to 120 per minute. The movements, together with the increase in number, are full; the intercostal and abdominal muscles being brought into play. The blowing respiration is accompanied by a pronounced back-and-forth body swaying. A clearing cough is likely to develop with a noticeable increase in the secretions of the mucous membrane of the nasal passages and presumably of those further back. Lacrimation is noticeably increased. Gradual recovery follows soon after the collapse. Without assistance the animal first regains a half dorsal position and soon rises, much depressed. The body-swaying and blowing respiration continue longer than the increase in the respiratory movements. The reaction may disappear within a few minutes or linger several hours. The animal may continue somewhat depressed for 24 hours following a decisive reaction. * * * An overwhelming dose however, will probably cause death by respiratory arrest.

In this work sensitization was produced by the initial dose of the foreign material, to which the animal reacted promptly on the administration of the second dose, and it serves as a shining example of the experimentally produced sensitization and anaphylaxis.

Holmes[3] published on "Anaphylaxis in the Larger Animals."

The first observation on induced hypersensibility to injections of alien proteins may be dated back to 1839, when Magendie found that rabbits which had tolerated two intravenous injections of egg albumin without any ill effects immediately succumbed to a further injection made after a few days.

Many theories have been formulated as an explanation of the phenomenon of anaphylaxis. Most authors agree that a specific antibody is formed in response to the primary injection. A great divergence of opinion arises on the question whether the agent which sensitized at the first injection is that which causes accidents after reinjection.

In agreement with this view that sensitization follows an initial injection of a foreign protein which is followed by anaphylaxis on the injection of a subsequent dose, we find ourselves at a loss to explain the occurrence of anaphylaxis in animals that are injected for the first time. Inasmuch as anaphylaxis is observed, it is generally assumed that these animals are naturally sensitive to the foreign protein injected for the first time. Why this sensitization occurs or in what percentage of animals it may be met with is not understood.

To have anaphylaxis develop in an animal that has received a prophylactic or therapeutic dose of a biological product, is both startling and embarrassing to the veterinarian, and quite difficult to explain to the owner. He invariably wants to know what could have been done to avoid the reaction or the fatality. It is, therefore, of basic importance that the administrator of a biological product concern himself with the nature of the material he is about to administer to the animal. He should at least note carefully whether or not a foreign serum is about to be used on a particular animal, as, for example, horse serum in the form of antianthrax serum in the simultaneous anthrax treatment for cattle.

In the employment of antianthrax serum (equine) in the simultaneous anthrax treatment of cattle, anaphylaxis has been noted, particularly in connection with the administration of the treatment in purebred horses. The development of anaphylaxis in such herds is apparently avoided in such instances in the use of antianthrax serum of bovine origin in the simultaneous anthrax treatment of the herd.

It is not always sufficient to rely on the use of a homologous serum to avoid the occasional development of anaphylaxis, inasmuch as it is becoming increasingly apparent that the alterations or changes that occur in a homologous serum heated to 58° or 59° C. for 30 minutes, with the subsequent addition of a phenolic preservative,

are more apt to be followed by the occurrence of anaphylaxis than occurs in the use of the same serum not so altered by the heating process. It, therefore, becomes of considerable importance in the production of antisera to preserve the sera without alteration such as caused by pasteurization procedures.

Since the pasteurization of animal sera is being advocated for the purpose of controlling virus infections, it is of importance to know whether or not such virus control can be accomplished with ways or means other than the pasteurization of the antisera. In this connection the observation is recorded that the aging of serum with phenolic preservatives over a definite period, approximately 30 days, will effectively destroy the infectivity of serum, including the virus of swamp fever. This result may be accepted as a definite indication that a definite time exposure on the preservation of antiserum will destroy such a virus as that of swamp fever, and that it is more than likely that other virus infections will succumb in a similar manner under such a time exposure. The present indications are that such a treated antiserum is less likely to be altered or denatured than one heated to 60° C. for 30 minutes.

Since pasteurized antisera have a wide distribution, and are employed extensively by practitioners throughout the United States, it is of importance that such sera be used with the caution that a certain percentage of animals will show anaphylactic reactions upon the initial use of such sera, regardless of whether the serum that is about to be injected is homologous or heterologous.

As to the procedure that might be followed so as to avoid the anaphylactic reaction in the employment of any antiserum, very little actually has been recorded on the attempts to determine which animals happen to be naturally sensitive to the antiserum about to be employed. It has been demonstrated clearly that sensitized animals can be shown to be sensitive by the use of a test dose of the serum employed by dropping a 1:10 dilution of the serum into the eye of the animal, or the injection of such dilution intradermally for the purpose of determining the local anaphylactic reaction.

Such a procedure may be of practical value to a very limited extent, where the circumstances are such that the procedure can be carried out to advantage. It is, however, unlikely that such a procedure can be put into general practice, inasmuch as such preliminary testing is not only time-consuming and costly, but at the same time economically unsound.

From a practical standpoint, therefore, we have but the following to adhere to, namely, that the animal may show an anaphylactic reaction on the initial administration of a foreign protein in the form of an antiserum, virus or bacterial vaccine; that such a reaction may prove fatal even though such an end result is rather unusual in spite of the alarming picture that the animal presents as the symptoms develop. The practitioner should give some consideration to the use of a homologous serum so as to reduce the incidence of anaphylaxis that occurs in the use of a heterologous serum.

While pasteurization of antiserum is practiced for the purpose of controlling the virus infections, such sera are likely to be accompanied by more anaphylactic reactions than unpasteurized sera. It may be well to give consideration to the use of aged, preserved sera for the purpose of controlling virus infections, so as to reduce the possibility of anaphylactic reaction in animals following the use of such sera.

The practitioner should be encouraged to make sensitization tests, particularly in those instances where the value of the animal and the conditions under which the serum is about to be used, would place the practitioner on the safe side of the procedure. It is further urged that the practitioner lend himself to attempts to pick out the sensitive animals, so that the value of such sensitization tests may be more clearly established in veterinary medicine.

REFERENCES

[1]Hoskins, H. P.: Anaphylaxis in veterinary medicine. Vet. Alum. Quar., vii (June, 1919), No. 1.
[2]Reichel, J., and Harkins, M. J.: Peptotoxin production by the bacillus of contagious abortion of cattle. Cent. f. Bakt., Orig., lxix (1913).
[3]Holmes, J. D.: Anaphylaxis in the larger animals. Mem. Dept. Agr., India, Vet. Ser., 1 (1913), p. 1.

(Discussion on page 442.)

EDITORIAL

The Journal

MEMBERS are urged to be patient and open-minded with our effort to "revamp the JOURNAL." The transformation is not the easy task it may seem. The Reorganizing Committee (Jakeman, Bergman, Brumley, Way), who have studied the needs of the Association in this and other respects, realized from the beginning the many ramifications involved in the reform and, therefore, worked slowly rather than to enforce revolutionary changes prematurely.

A periodical is the voice of its readers. It reflects their thoughts, their ideas, their knowledge and their labors. Since our profession is composed of a number of branches somewhat unrelated in their application, covering the entire scope to the pleasure of all concerned is a baffling undertaking—baffling because the Association as managed through its 76 years of life has a limited income which governs the amount of white space it can afford to fill up with printed matter once a month. So, while a better and bigger JOURNAL is being built up along with a better and bigger membership and while the accounting department is keeping all hands within sensible limits, the plea for patience is not unreasonable.

The general plan is to create new departments which will portray the passing events of veterinary medicine as accurately and as completely as possible and to do this as fast as the central office and the nonresident associates can be organized into a staff of editors charged with keeping the technical and professional news of their specialties up to date.

This office has a wealth of scientific papers awaiting publication but, since the majority of them comes from authors in high places, they are mainly of a highly technical nature, reporting the kind of fine, original work that has lifted the veterinary profession to a higher level in this country but which, on the other hand, are interspersed with lengthy details of interest only to the few belonging to the given branch. To somehow find room for these contributions is our obligation to the authors and to work out means of doing so is our duty to the profession. But whether this should be done at the expense of the JOURNAL'S general popularity is the question to be answered. Letters in large numbers received from members in key positions and in all branches replying to the direct question, "What should be done to make the Association more useful?" nearly all recommend that changes be made in the contents of the JOURNAL. Specific advice that can be used forthwith is, however, still lacking. The danger of offending one branch and slighting another remains extant, and will always remain so until the membership is enlarged and funds to carry on with more printed pages, slighting no branch, become available as a consequence of the increment.

The A. V. M. A. is not poor. In the manner of speaking it "has money in the bank," and stands upon a solid financial foundation, but in longtime planning it must keep its budget in balance. Either the membership must be built up or the dues increased. The former is feasible and should be accomplished in order that the latter does not become a financial necessity. This is not the place to argue the importance of organized veterinary medicine. Very few non-members read these pages. The obligation to tell the story rests upon the leaders of the different branches, the state secretaries and the personnel of this office, who must somehow make a membership worthwhile.

This plea for patience includes a request that authors consider space a matter of importance just now by contracting con-

texts and tables and appending summaries to articles as a guide to the mass of readers not momentarily interested in the details. This hint alone would open space for the matters of current interest which, according to popular gossip, is the practitioners' reason for not joining the Association. The practitioner potential is not fiction. It comprises an army of many thousand whose annual dues would enable the JOURNAL to broaden and the Association to carry out the important projects it is undertaking.

In short, all surveys of the perspective from the central office lead to the same conclusion—enlarge the membership and spend the money for its benefit.

The Age of the Journal

Expressed in the usual Romantic numerals, the JOURNAL will enter its XCVth volume with the July, 1939, issue. It was founded in 1877, or 62 years ago, but the forthcoming volume will be the 95th instead of the 63rd because there have been the semi-annual volumes to add to the confusion, not to mention the starting of a "New Series" when the Association for the second time took over the publication in 1915, and did the unusual thing of starting a new series of birthdays, instead of building upon the most precious possession a periodical can boast about—its age.

The confusion can never be entirely overcome but much of it can be removed by abolishing the "New Series" idea and going back to the more dignified plan of using but one figure, the figure giving the number of volumes actually published, which would make the coming volume number XCV (95), and thus allow the "new series figures" to pass into oblivion as an unfortunate departure from the conventionalities of periodical publications.

Those who notice details are aware that the old number alone appears on the backbone of current issues with the name of the JOURNAL and pages an issue covers, in order that issues and pages can be easily identified when stacked on the shelves of a library. Beginning with this issue a running head will show the date on each page, which was a strange omission of the past.

Suggestions on this matter are invited, as there is no intention to make important changes without the consent of the membership.

Convention Plans

Are you making your plans to be in Memphis the last week in August? The Committee on Local Arrangements is now completely organized and is holding regular monthly meetings for the purpose of developing plans for the 76th annual meeting of the A. V. M. A. The Peabody Hotel will be official headquarters. Monday, August 28, will be the first day of the convention, officially, and will be given over to meetings of the Executive Board, committee meetings, and the first session of the House of Representatives. The opening session of the convention will be held on Tuesday morning, August 29. Sectional meetings will consume Tuesday afternoon, all day Wednesday and Thursday morning. Thursday afternoon and evening will be given over to entertainment and, on Friday, an all-day clinic will be held.

Alert section officers are already compiling their programs which, according to reports arriving, will make up an intellectual entertainment overflowing with material of the first rank; and the program for the general session is well under way, thanks to their spirited coöperation with the central office.

The exhibit booths were "snapped up" within three days after the applications were mailed to the prospective exhibitors. However, steps are being taken by the Committee on Local Arrangements to provide satisfactory space for exhibitors whose applications arrived too late to be assigned to the regular exhibit room.

The interest that is being shown in the 76th annual meeting is a wholesome forecast of a great convention.

The committee on educational exhibits has sent invitations to the veterinary col-

leges in the United States and Canada offering them the opportunity to prepare an exhibit or display representing their school. An excellent room has been set aside for the educational exhibit and some very interesting specimens will be shown.

On the Shortage of Veterinarians

Those who keep informed on the political situation in the principal countries of this period will agree that more attention than formerly is given to the conservation of live stock. The aim is to become self-supporting in the matter of food supply and, in making surveys to that end, the veterinary service has loomed up suddenly to many statesmen as a factor of the first rank. The animal population must somehow be improved in kind and number and, logically, men of veterinary medicine are looked to for intelligent aid and advice. Overlooked by the masses during two centuries of historic developments in the liberal arts and sciences, the care of domestic animals is being appraised at its par value.

In defending a proposed law before the Chamber of Deputies of France on regulating (within stated limitations) the practice of veterinary medicine by non-graduates, Deputy Blanchoin said:

> The problem before the Chamber is dominated by the following figures: There are 40 million head of live stock in France and only 2,400 graduate veterinarians, while in Germany, to cite but one example, there are 7,000 veterinarians for 28 million of such animals. *Under such a state of affairs, can we dispose of 8,000 non-graduates without doing mortal injury to our animal industry?* No! Not as long as we remain faithful to the policy of the "*numerus clausus*" that arbitrarily keeps the number of graduates below the needs of agriculture.

The veterinary schools of Lyon, Alfort and Toulouse, are the "*numerus clausus*" pointed out, which limit by competitive examination the number of students to be admitted and, thereby, they allow the development of a large cohort of expert blacksmiths and castrators (*maréchaux-experts et hongreurs*), who seek license to fill the gap that the formal educational system failed to close with the growth of veterinary science to a status of greater importance along with industrial development, improved standards of living, international strife, war and threats of war.

In years gone by, the veterinarians of France had not been perturbed by the army of *maréchaux-experts et hongreurs*, the equivalent of our licensed and non-licensed non-graduates. On the contrary, precisely as other countries, including our own, have done, a part of the veterinary service was permitted without protest to fall into outside hands. Licensure laws were not thought to be necessary as protection for graduates. But now that a wider comprehensibility of the relation of live stock to human welfare has gripped the minds of statesmen, the graduates of veterinary colleges are found to be too sparse to make up a sufficient disease-fighting front without filling the ranks with dubious recruits, devoid of conventional training and ethical conduct.

The problem is one for the A. V. M. A. to ponder. We are trying to publicize our profession with the object of expanding its field without complementary plans for supplying a sufficient number of college graduates to avert the coming of the evil hour when statesmen will find it expedient to muster incompetent practitioners into the service and specialists to direct them, while the meaning of D. V. M. remains undefined in the glossary of the political circle. Or, are we not already *vis-à-vis* a comparable condition?

President Bergman will attend California meetings in June as a part of his extensive presidential itinerary and thus round out a year of extraordinary contributions to the cause of veterinary medicine. His visits to veterinary and other scientific societies are events that will long be remembered.

The Reorganization Committee (Jakeman, Bergman, Brumley) met in Chicago February 17-18, 1939, when they mapped out the major projects of the future which grew out of the third year of intensive study of the Association's affairs.

The Shute Test for Vitamin E Deficiency

Abderhalden[1] first clearly demonstrated that the serum from pregnant animals behaved abnormally in respect to its antitryptic activity. The test, however, failed as a specific diagnostic for pregnancy, since male animals frequently showed positive reactions, pregnant females sometimes showed negative, and positive reactions were obtained also in states of malignant cachexia, in acute infections, in neural imbalances and mental states such as dementia praecox and in certain forms of luetic infection. All that remained of the Abderhalden test was the evidence that the blood serum is altered frequently in pregnancy and in other conditions, but the significance of the alteration wes unexplained until recently.

Shute,[2-4] following the work of Fine[5] and others, has reversed the Abderhalden procedure. Instead of measuring the antitryptic action of blood serum, Shute measures the digestibility of blood serum by trypsin. From observations on 1,200 humans, male and female, and on laboratory animals, Shute concludes that his test apparently reveals a high and abnormal level of estrogenic substances in the blood such as may accompany vitamin E deficiency.

These findings are amazingly interesting and seemingly clarify the nonspecificity of the Abderhalden test. A gestation course, during which the vitamin E utilization of the body is impaired such as to threaten abortion, shows a positive Shute test and a positive Abderhalden; but if abortion is not threatened, implying vitamin E sufficiency, the Shute test remains negative as does the Abderhalden. Thus, both tests agree in revealing a state resulting from vitamin E deficiency and not merely pregnancy.

Davidson[6, 7] has shown that vitamin E surfeited white mice do not tend to develop inoculable cancer, suggesting the restraint of lawless cell growth because of inhibition by vitamin E. Adamstone[8] has shown that in the absence of vitamin E, chicks develop a malignant form of cancer akin to the prevalently disastrous fowl leukemia. Thus, the prevention of cancer in mice, in proportion as the vitamin E intake is raised, and the development of cancer in poultry when the vitamin E supply is diminished or extinguished entirely, suggests strongly that mass cell reproduction, like the somatic reproduction of pregnancy, involves vitamin E participation; and when this is aberrant, once more the Abderhalden test reveals the insufficiency. It is proper to suggest, then, that positive Abderhalden tests in cancer cases are explainable if interpreted in the light of the Shute phenomenon.

In cattle, acutely infected Bang's disease reactors have so far given exclusively positive Shute tests. Also, a positive reactor showing at the same time a positive Shute test has invariably aborted. Contrariwise, there is now a record of one pregnant cow that became acutely infected with Bang's disease and gave a strongly positive Shute test. At the fifth month of pregnancy, when the abortifacient epidemic broke out in the herd, which had been clean for three years previously, this cow was saturated with wheat-germ oil, and the saturation repeated each week thereafter. The reaction to Bang's disease is still positive. The Shute test became negative after the second saturation, and the cow delivered an apparently normal calf at full term which is alive and vigorous now at two months of age. It will be recalled that Abderhalden and Andryewsky[9] obtained positive tests with cattle which they acutely infected with tubercle suspensions, so that infections more closely related to reproductive disorders and perhaps associated with vitamin E deficiency, as Moussu's[10] work seemingly implies for infectious abortion, may similarly be expected to show the Shute phenomenon. This is confirmed in the case of the single animal mentioned.

Einarson and Ringsted[11] have produced muscular and spinal cord lesions in adult rats by vitamin E depletion. They state that the pathological-anatomical changes

which they have experimentally produced resemble two of the most important conditions in man, namely, tabes dorsalis and spinal progressive muscular atrophy. Tabes may arise, therefore, from vitamin E depletion, or vitamin E depletion may so undermine the integrity of the nervous system as to make it more readily invaded by syphilis. Here again, the Abderhalden test probably measures vitamin E deficiency, explainable according to Shute.

From Shute's work there emerges a reasonable reconstruction of a newer interpretation of the Abderhalden test. It is not a specific test for pregnancy, as all agree, but it is seemingly a test for vitamin E deficiency however produced, in males and in females, in abnormal gestations, in acute infections, in luetic invasions and in the cachexias of malignancy.

Shute thus furnishes a potential test that should be of inestimable value to the veterinarian. From his experiments and observations on numerous humans, he has reached conclusions that shatter the unsustained premises academically heralded about vitamin E. Shute seems to have shown that: Vitamin E deficiency is quite widespread, it being not at all uncommon to find seven out of eight individuals with positive Shute tests; indeed, vitamin E deficiency is often so pronounced that large initial quantities of wheat-germ oil are required to "saturate" the individual; green feeds have been extolled too highly as sources of vitamin E, such feeds having most generally failed to replace wheat-germ oil for the correction of pregnancy disorders in the human; animals respond to the test as do humans.

The extension of the latter hypothesis of Shute, that animals respond to the Shute test as do humans, is clearly the investigative province of the veterinarian. It is possible, of course, that the Shute test may need to be modified, or even exchanged for the Aberhalden test which is its serological progenitor, to make it more applicable to the differences which distinguish the blood of animals from that of humans. Those who have so far employed the test with cattle, dogs and other animals see the possibility of applying such a test to aid in the study and eventual correction of the vast and extensively scattered reproductive failures that are everywhere undermining the profitable progress of live stock and poultry development—a huge economic loss for which vitamin E deficiency is in a very large measure assuming definite responsibility.

REFERENCES

[1]Quoted by Kolmer, J. A.: Infection, Immunity and Biologic Therapy. (W. B. Saunders Co., Philadelphia, 1925.)

[2]Shute, Evan: Resistance to proteolysis found in the blood serum of aborting women. Jour. Obstet. & Gynaecol. (Eng.), xlii (1936), 6, p. 1071.

[3]Shute, E.: Relation of deficiency of vitamin E to the anti-proteolytic factor found in the serum of aborting women. Jour. Obstet. & Gynaecol. (Eng.), xliii (1936), 1, p. 74.

[4]Shute, E.: Determination of the estrogenic substance in blood serum by means of an estimation of the anti-proteolytic power of the serum. Amer. Jour. Obstet. & Gynaecol., xxxv (1938), 6, p. 970.

[5]Fine, J.: On the influence of serum on enzymes with special reference to its action on trypsin. Biochem. Jour., p. 647 (1931).

[6]Davidson, J. R.: An attempt to inhibit the development of tar carsinoma in mice. Can. Med. Asso. Jour., xxxi (1934), p. 486.

[7]Davidson, J. R.: An attempt to inhibit the development of tar carsinoma in mice. Can. Med. Asso. Jour., xxxi (1934), p. 486.

[8]Adamstone, F. B.: A lymphoblastoma occurring in your young chicks reared on a diet treated with ferric chloride to destroy vitamin E. Amer. Jour. Cancer, xxviii (1936), 3, p. 540.

[9]Abderhalden and Andryewsky: Munchener Tierärzt Wchnschr., lxi (1913), p. 1641.

[10]Moussu, R.: Sur l'avortement epizootique des Bovides. Rec. de Méd. Vét., cxi (1935), p. 905.

[11]Einarson, L., and Ringsted, A.: Effect of chronic vitamin E deficiency on the nervous system and the skeletal musculature in adult rats. (Oxford University Press, London, 1938.)

Correspondents

The central office will deem it a favor if correspondents will address all communication to the "American Veterinary Medical Association," 221 North LaSalle street, Chicago, rather than to any person connected therewith. Personalities may be left to the letter contained. The object is to build up the name of the Association rather than the name of any one it happens to employ. Men pass on but the Association goes on forever. The new stationery of this office is impersonal but this does not mean that correspondents should not call us by our first names in the letter itself.

The Poultry-Practice Situation

It seems unnecessary to point out that veterinarians in some places are not making the best of the situation presented by the tremendous development of the poultry industry and the high incidence of disease among that class of farm animals. It is our belief, however, that poultry practice will develop only as fast as veterinarians qualify in the handling of avian diseases. The interest in this branch of medicine will always remain subordinate to that factor.

It seems to us that lack of interest in avian medicine is more imaginary than real and that veterinarians are slow in building up a clientele among poultrymen (which, in effect, also means the farmers' wives), because definite plans of utilizing avian medicine economically have yet to be worked out. Perhaps too little attention has been paid to the planning of ways to make correct diagnoses and too much to the furnishing of hit-or-miss remedies which, in the absence of diagnoses, are only duplications of the nostrums sold through various channels.

The following is a quotation from a letter we received on this important question of poultry practice:

> It is difficult to arouse the interest of veterinarians in poultry practice. They fail to realize the value of poultry and poultry products on the farms in their state or immediate territory and, on this account, are more or less blinded to the enormous amount of money going out of each locality every year to peddlers of various poultry remedies. A great deal of this could be saved the poultry producer, and a very ethical and profitable enterprise may be worked out by the local veterinarian, if he will avail himself of existing information on poultry husbandry, disease, and sanitation.
>
> In my experience, too few veterinarians have any knowledge of poultry husbandry and, as far as diseases are concerned, they may carry a certain line of remedies purchased through commercial houses. These are dispensed in many instances without a definite diagnosis. The results, of course, are very erratic and, to the producer in many cases, discouraging. The same man would fail in practice if he conducted his business similarly with other species of live stock.
>
> In many localities, there is too much animosity between hatcheries and veterinarians. This should be overcome. This may be difficult or impossible in all cases, and I certainly do not favor many hatchery programs. In many instances where this coöperation has been established, it has been profitable for both parties. Holding disease clinics, by a veterinary group in a county, through coöperation with the federal and state veterinarians, and possibly the county agent and hatcheries, has been one way to bring all parties together and to a better understanding of each phase of the industry. Producers have demonstrated by their purchases each year that they are willing to pay for service. This service might, and should, be rendered by veterinarians.
>
> These are merely some of my observations in this state during the past year, where we have been attempting to wake up the profession to the realization that a great deal of business that rightfully should be theirs is going to someone else.

It was stated that the lack of interest in avian medicine shown by practitioners is more imaginary than real, because veterinary poultry conferences, conducted by various universities, have always attracted good audiences keenly interested in mastering the subject.

Everyone will recall the confusion that once surrounded the development of swine practice. Now, history is repeating itself in the case of poultry practice. Every veterinarian should take a correct inventory of the live stock and poultry population of the district in which he practices. He should qualify in the handling of the diseases of each species and, thus, turn all owners of animals towards their veterinarian when disease occur. Obviously, owners of animals will seek advice and treatment where the best solutions of their problems can be obtained.

With the new regulations of the Food and Drug Administration in effect,* the advantage will be all on the side of scientific avian medicine.

*Journal of the A. V. M. A., February, 1939, pp. 75-77.

Cornell University holds a short course on farming, nutrition and health for missionaries, to help them aid the community to which they are assigned.

Election of Section Officers

Inasmuch as there has been some debate in the several sections on the important question of choosing their officers and other reforms, it seems advisable to state at this time that the election of the officer for a coming year was discontinued because in the enthusiasm of a meeting, they were not always wisely chosen in regard to their ability to serve the section to the best advantage. In several instances, officers elected at meetings did not take the keen interest in the work of the section required to do justice to their branch and in not a few instances, the elected officers did not attend the meeting at all, leaving the section without a head for that year.

Faced with these facts, the Executive Board decided that presidential appointments would be preferable for the reason that the President would naturally confer with the leaders of the given section.

Executive Board Elections

The regular elections for members of the Executive Board in Districts 4 and 10 have been in progress since February 28. Members of the A. V. M. A. in these two districts have been casting their nominating votes in the primary elections, which will come to a close on April 28. As soon as possible after that date, the ballots for the elections proper will be prepared and mailed to all paid-up members in the two districts. The states of Kentucky, Virginia, West Virginia, Maryland, District of Columbia, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Florida, Cuba, West Indies, and South America constitute District 4, and the states of Ohio and Michigan constitute District 10. The polls for the elections proper will remain open until June 28, 1939.

If you have not received a ballot, perhaps your dues have not been paid. Check up on this.

Way on His Way

President-Elect Way, accompanied by Mrs. Way, passed through Chicago, Sunday, March 19, on his homeward trip to New York from California, where he had spent several weeks of mixed business and pleasure recuperating from the serious illness which followed a major operation last December.

Between trains he conferred with officials of the Association on the gigantic publicity campaign he planned and sponsored at the New York meeting and which is being continued under his direction through the current year.

Members and non-members are reminded that the Executive Board has placed $2,000 at his disposal for the purpose of doing the patriotic duty of telling the story of the veterinary service to the people and thus aid in removing some of the obstacles under which the veterinarian labors.

Foot-and-Mouth Disease

During our recent European trip, information was obtained to the effect that foot-and-mouth disease in a foreign country across the sea resulted from the administration of a contaminated biologic and it is now mentioned in the *Canadian Journal of Comparative Medicine.** This finding emphasizes the need for watchfulness over all products proceeding from infected countries, if introduction of the disease is to be prevented.

A cow that had been given an injection of pituitary extracts in an effort to induce estrus exhibited symptoms of foot-and-mouth disease 48 hours after treatment. Cattle and guinea pigs were given injections of extract of the same serial number and it was established that all of the doses administered were infected with the virus of that disease. The product had been imported from a continental country where there has been widespread existence of foot-and-mouth disease during the past 18 months.

J. R. M.

*Vol. 3, No. 1, January, 1939, p. 23.

APPLICATIONS

The work of the resident state secretaries and other officials during 1939 is reflected in the gradual increase in the number of new members listed from month to month in this column—the barometer of A. V. M. A. popularity. But considering that there are still from six to seven thousand eligibles to be brought into the membership roll, much remains to be done to make the Association the universal "lodge" of the veterinary profession, and thus expand its influence.

First Listing

(See January, 1939, JOURNAL)

CADY, LT. DUANE L.
Veterinary Station Hospital, Fort Snelling, Minn.
D. V. M., Kansas State College, 1934. Vouchers: Joab P. Foster and J. G. Fuller.

CAIN, WILLIAM
3328 S. Claiborne Ave., New Orleans, La.
D. V. M., U. S. College of Veterinary Surgeons, 1921. Vouchers: W. A. McDonald and D. D. Conner.

COCHRAN, JOE ROBINSON
533 Tuscaloosa Ave., Birmingham, Ala.
D. V. M., Alabama Polytechnic Institute, 1935. Vouchers: McKenzie Heath and R. L. Mundhenk.

COOK, RAYMOND W.
Pine Road, Fox Chase, Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1933. Vouchers: Allen S. Vansant and Joseph W. Vansant.

COOK, ROBERT WILLIAM
710 Bradley Ave., Peoria, Ill.
D. V. M., Kansas State College, 1936. Vouchers: C. L. McGinnis and Bentley F. Hudson.

DAVIS, DAVID EVERETT
176 Main St., Petaluma, Calif.
D. V. M., Kansas State College, 1922. Vouchers: C. M. Haring and J. R. Beach.

DEESE, LESTER ARVEL
Duke Inn, Elizabeth City, N. C.
D. V. M., Alabama Polytechnic Institute, 1938. Vouchers: J. W. Woods and Wm. Moore.

EGGERT, WILLIAM E. JR.
Office of the Corps Area Veterinarian, Governors Island, N. Y.
D. V. M., Cornell University, 1935. Vouchers: John B. Hopper and C. W. Greenlee.

HANCOCK, EDWARD ERROL IRWIN
Provincial Animal Pathologist, Truro, Nova Scotia, Can.
B. V. Sc., Ontario Veterinary College, 1924. Vouchers: Thomas Moore and F. A. Humphreys.

HANSON, JOHN CLARENCE
34 W. Central St., Chippewa Falls, Wis.
D. V. M., McKillip Veterinary College, 1915. Vouchers: James C. Green and James S. Healy.

HARLAN, LT. WILLIAM HENRY
1916 N. W. 38th St., Oklahoma City, Okla.
D. V. M., Colorado State College, 1929. Vouchers: S. E. Douglas and L. J. Allen.

HOBBS, JOSEPH WILLIAM
1004 Second Ave., Selma, Ala.
D. V. M., Alabama Polytechnic Institute, 1938. Vouchers: R. L. Mundhenk and F. P. Woolf.

HOLLOWAY, ALBERT LEE
Atmore, Ala.
D. V. M., Alabama Polytechnic Institute, 1921. Vouchers: R. L. Mundhenk and F. P. Woolf.

JENKINS, ORVILLE B.
330 Federal Bldg., Madison, Wis.
D. V. M., Kansas City Veterinary College, 1913. Vouchers: James S. Healy and William R. Winner.

JONES, THOMAS BENTON
2142 W. Van Buren St., Phoenix, Ariz.
D. V. S., Kansas City Veterinary College, 1907. Vouchers: S. E. Douglas and Ward R. Lee.

LARSON, RAYMOND EDWARD
Towson Veterinary Hospital, Towson, Md.
V. M. D., University of Pennsylvania, 1938. Vouchers: John D. Gadd and Harry A. Meisner.

MCCARTY, FRANCIS A.
Rosendale, Wis.
D. V. M., McKillip Veterinary College, 1918. Vouchers: James S. Healy and William R. Winner.

MAINHART, CHARLES HORINE
350 Woodland Ave., Richmond, Ky.
M. D. V., McKillip Veterinary College, 1906. Vouchers: Floyd E. Hull and W. W. Dimock.

MAURER, E. LAVERNE
304 Federal Bldg., Great Falls, Mont.
B. S., D. V. M., State College of Washington, 1938. Vouchers: W. L. Carson and G. W. Cronen.

NAGLE, ALBERT C.
Box 174, Harrisburg, Pa.
D. V. M., Michigan State College, 1938. Vouchers: S. A. Peck and Ward Giltner.

Reichert, Paul F.
Route 2, Chelsea, Mich.
D. V. M., Michigan State College, 1938. Vouchers: Lloyd B. Sholl and C. F. Clark.

Ritter, Earl Conrad
Sumner, Iowa
D. V. M., Iowa State College, 1938. Vouchers: F. F. Meads and C. A. Stewart.

Safford, John W.
Box 129, Lewiston, Idaho.
B. S., D. V. M., Washington State College, 1938. Vouchers: E. E. Wegner and J. E. McCoy.

Schneider, Morris D.
Box 154, Boscobel, Wis.
D. V. M., Alabama Polytechnic Institute, 1936. Vouchers: James S. Healy and William R. Winner.

Schwartz, Albert V., Jr.
326 Post Office Bldg., Baton Rouge, La.
D. V. M., Kansas State College, 1938. Vouchers: Richard E. Omohundro and W. A. McDonald.

Scrivner, Lloyd Herbert
Cornell University, Ithaca, N. Y.
D. V. M., Colorado State College, 1929. Vouchers: Aubrey M. Lee and Clifford W. Barber.

Shelby, Clarence Franklin
326 Post Office Bldg., Baton Rouge, La.
D. V. M., Kansas State College, 1938. Vouchers: Wilbur H. Wiswell and L. A. Merillat.

Strandberg, H. L.
Glenwood, Minn.
D. V. M., Iowa State College, 1931. Vouchers: E. H. Gloss and Carl Hansen.

Terry, Tomas A.
Calle A esquina a 21, Vedado, Havana.
V. D., University of Havana, 1938. Vouchers: Bernardo J. Crespo and L. A. Merillat.

Theophilus, Donald K.
Fort McPherson, Ga.
D. V. M., Iowa State College, 1936. Vouchers: Col. B. A. Seeley and J. C. Wright.

Tracey, Richard W.
Parkton, Md.
V. M. D., University of Pennsylvania, 1932. Vouchers: Harry A. Meisner and John D. Gadd.

Van Sant, Willard Merrill
824 E. State St., Boise, Idaho.
D. V. M., Kansas State College, 1937. Vouchers: Bernard I. Copple and A. J. Creely.

Weisner, Ernest Steven
1009 West Grand River Ave., East Lansing, Mich.
D. V. M., Michigan State College, 1937. Vouchers: E. E. Hamann and H. J. Stafseth.

Whitehead, Lt. Charles Joseph
2711 S. 13th St., Tacoma, Wash.
B. S., D. V. M., Washington State College, 1938. Vouchers: Col. R. J. Foster and Lt. Col. S. C. Dildine.

Wilkin, William Arthur
413 S. Sycamore St., Centralia, Ill.
D. V. M., Chicago Veterinary College, 1915. Vouchers: C. C. Hastings and W. B. Holmes.

Ziebell, Vernon Frank
Tomah, Wis.
D. V. M., McKillip Veterinary College, 1918. Vouchers: James S. Healy and William R. Winner.

Zinober, Moses Robert
602 Lincoln St., Antigo, Wis.
D. V. M., Michigan State College, 1938. Vouchers: James S. Healy and William R. Winner.

Second Listing

Alcorn, Linden Moore, 336 Post Office Bldg., Oklahoma City, Okla.
Anderson, Walter A., 308 Federal Office Bldg., Seattle, Wash.
Berry, Thomas C., Auburn, Ala.
Bohannon, Vincent Denhard, 2506 W. Linden Ave., Nashville, Tenn.
Chadwick, Charles William, 805 Florida Court, Gainesville, Fla.
Champlain, Lloyd, Cinema, B. C., Canada.
Dean, William Douglas, Box 98, Auburn, Ala.
de Hall, John Cornelius, Jr., 1701-16th Ave. S., Birmingham, Ala.
Dovre, Odin E., Box 357, Tupelo, Miss.
Etheridge, Joseph W., 1016 Fenwick Lane, Silver Spring, Md.
Gross, William F., Box 2315, Cristobal, Canal Zone.
Harz, Hubert H., 1454 Pensacola Ave., Chicago, Ill.
Henderson, James Arnold, 36 Broad St., Flemington, N. J.
Hervey, Lt. William Hugh, Office of the District Veterinarian, Fort Leavenworth, Kan.
Hofferd, Raymond Mathias, 2311 B. Avenue, N. E., Cedar Rapids, Iowa.
Isa, Jay Meyer, University of Manitoba, Winnipeg, Man., Canada.
Mathis, Rudy Cletus, Laurel, Miss.
Mogge, Arthur Theodore, Eskridge, Kan.
Morris, Alexander, 85-11 167th St., Jamaica, N. Y.
Peters, Stanley Edward, General Delivery, Fayetteville, Tenn.
Randall, Charles Bernard, Box 262, Snow Hill, N. C.
Rohrer, Raymond R., Orwigsburg, Pa.
See, John Newlove, Route 4, Malton, Ont., Canada.
Severn, S. S., Box 141 Seguin, Texas.
Shaw, Charles B. 743 Main St., Leominster, Mass.
Smith, Robert P., Jerome, Idaho.
Spong, Lawrence Eric, Box 96, Iola, Kan.
Stroup, W. L., Corinth, Miss.
White, Edward S., 1102 State Office Bldg., Richmond, Va.

The amount which should accompany an application filed this month is $8.75, which covers membership fee and dues to January 1, 1940, including subscription to the Journal.

CLINICAL DATA

Tibial Fracture in a Horse Treated Successfully*

By J. F. THOMAS, *Oswego, Kan.*

Late in the evening of August 31, 1938, accompanied by my assistant, Wayne Collins, and my son — senior and sophomore, respectively, of the Kansas State College— I planned a successful treatment of a horse with a broken leg. The horse, Wishbone, is a valuable seven-year-old Palomino stallion, formerly owned by the late, lamented Will Rogers and now the property of the Goodwin ranch, southeast of Coffeyville, Kan. The animal had just returned from leading a parade, where he had suffered a fracture of the right tibia, caused by a sudden twist of his body as the saddle fell under him.

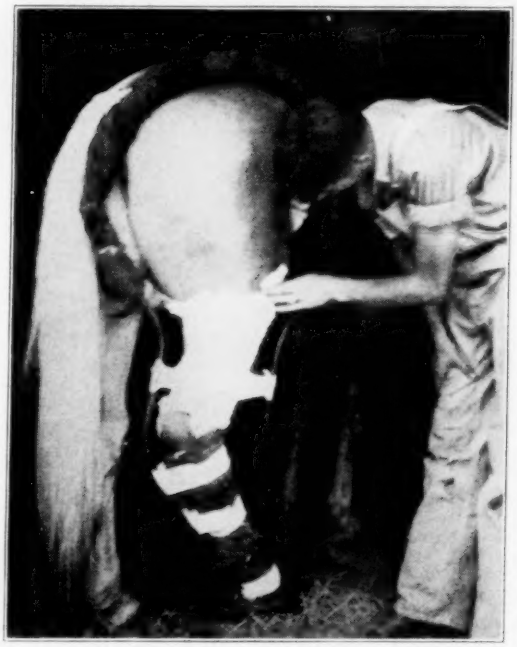


Fig. 1. Splint being applied.

The injury was a simple oblique fracture located at the middle of the bone. The seriousness of the injury was explained to the owners. They were willing to make every effort to save the animal, since he was valuable for breeding purposes.

The patient was placed in slings. A pattern for a splint was fashioned with No. 9 wire and taken to a blacksmith, who made a Thomas splint of ⅜-inch gas pipe. The leg was bathed with hot and cold packs alternately to remove the swelling and, on the following morning, the splint was applied (fig. 1), using the technic described below.

The leg was well padded with long-fiber cotton. Light-weight board splints were applied and held in place with three-inch gauze bandages reinforced with two-inch wet-proof tape. The metal splint was padded to fit snugly about the medial part of the thigh. Fortunately, since the animal was shod, the distal end of the splint could be fastened by means of wire to the shoe. By exerting pressure proximally and tension distally, the bones were held as immobile as possible. The weight of the splint was partially supported from above by means of a rubber innertube.

In about twelve days, considerable swelling and suppuration developed from the pressure of the wooden splints. These were removed and the infected area was drained by a bold incision. The ends of the bone could be felt in the wound. The fracture now being compound, a complete recovery seemed impossible, unless a different type of support could be devised. This was accomplished in the following manner.

*Received for publication, January 9, 1939.

The fracture was supported medially by a piece of four-inch, well padded strap iron anchored anteriorly and posteriorly to the gas-pipe splint. Laterally, the fracture was supported by a 1″ x 6″ board, attached in a similar manner. A short piece of 1″ x 4″ board was inserted posteriorly to hold the hock in position. In other words, the fracture was incased in a box-like structure. It is obvious that the purpose of the second modification was to prevent the sideward movement of the bone and, at the same time, not interfere with the normal circulation.

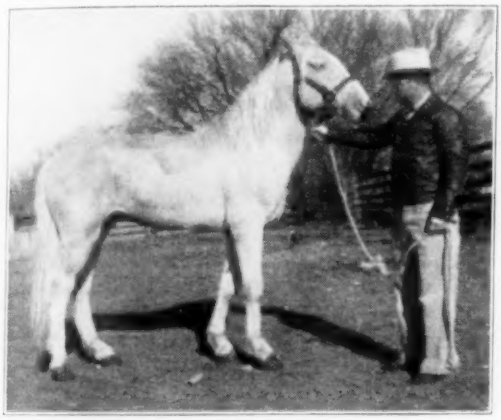


Fig. 2. Patient, four months after injury, apparently completely recovered.

Seventy-two days after the leg was set, the splints were removed. The fracture appeared to be mended with a considerable amount of bone formation. The animal was kept in the slings for an additional two weeks, without exercise. The two weeks following, the patient was exercised moderately and returned to the slings for rest. It is now four months since the break and, at the present time, the horse seems to have made a complete recovery (fig. 2). Two injections of 1,500 units of tetanus antitoxin were given 60 days apart and calcium gluconate powder was added to the feed during the period of treatment.

The journalist should be on his guard against publishing what is false or exceptionable in morals. — *William Cullen Bryant.*

Marsh's Disease*

By A. SAVAGE *and* J. M. ISA, *Winnipeg, Man., Can.*

Veterinary Laboratory, Manitoba Department of Agriculture

In affixing the name "Marsh's disease" to the disease herein discussed, we may seem rather bold, since the U. S. Bureau of Animal Industry has called it "X." To the best of our knowledge, however, Marsh[1, 2] was the first to describe the disease systematically. It seems, therefore, that Marsh is as much entitled to have this entity associated with his name as were Hodgkins and Johne with the entities they described.

In 1937, Marsh described a peculiar disease of horses that, in his experience, followed the use of an anti-encephalomyelitis serum more often than not. For the past two years at least, the same disease has occurred in Manitoba, where it was unnoticed prior to the rather widespread use of the antiserum mentioned.

Recently we have had occasion to examine a volume of material in accordance with the method described lately by Green and Evans[3] for the purpose of determining the presence of cellular inclusions in cases of suspected canine distemper. In consequence, we were subconsciously on the alert for them. Therefore, it was perhaps by coincidence that we found somewhat similar bodies in sections of liver prepared from local cases of Marsh's disease. These inclusions also were noted in material forwarded by Dr. Marsh for comparison.

The bodies in question are virtually spherical. Their size varies from 2μ to approximately 15μ in diameter. In some cases they are surrounded by clear zones or haloes. Almost without exception there is only one in a liver cell, although according to our observations, in very few cases are there any at all. In badly degenerated portions of the liver they may persist in a free state for some time after the mother cells have disappeared. Eventually they disintegrate.

Sections stained with haematoxylin and eosin show the inclusions to be acidophile.

*Received for publication, March 15, 1939.

When Wright's stain is used, they are often less red than the surrounding cytoplasm. Under high aperture (1.30), they are not entirely structureless. Thus far, however, we have been unable to demonstrate that they contain chromatin.

In the absence of experimental work and of more widespread observations, we express no opinion as to their cause and significance. However, in view of the fact that similar objects commonly accompany or result from virus infection, the existence of these inclusions seems to be worth recording.

REFERENCES

[1]Marsh, H.: Jour. A. V. M. A., xci (1937), n. s. 44 (1), p. 88.

[2]Marsh, H.: Jour. A. V. M. A., xci (1937), n. s. 44 (3), p. 330.

[3]Green, R. G., and Evans, C. A.: Corn. Vet., xxix (1939), 1, p. 35.

Fetal Ascites*

By J. MICUDA, *Gaithersburg, Md.*

Recently, I encountered a case of dystokia which seems worthy of report. The subject, a six-year-old Guernsey cow, was standing in a stanchion in the milking barn. Protruding from the vulva were the front feet and head of a calf. Ropes had been tied to the feet and it was evident that traction had been applied. The calf was dead.

Vaginal examination revealed a normal presentation but a greatly distended abdomen. Fortunately, the only thing I could think of was ascites. I removed the head of the fetus, which apparently had gone full term and, with an obstetrical finger knife, I made an incision in the flank of the calf just behind the last rib. A quantity of reddish fluid spurted from the opening and flowed out through the vulva. The abdominal viscera was then removed and more fluid gushed out. Finally, with a little manual traction, the fetus, a male, came away. The scrotum was about the size of a quart milk bottle from the fluid. There were between five and six gallons of fluid in the abdomen of the fetus. The diaphragm was not ruptured and the fetus was not abnormal in any other way. The abdominal cavity held all of the fluid. After deflation, the fetus weighed about 75 or 100 pounds.

Previously, the cow had given birth to three normal calves.

*Received for publication, January 5, 1939.

Trout Poisoning*

By A. M. MCCAPES, *California Polytechnic School, San Luis Obispo, Calif.*

During the recent steelhead-fishing season in California, I was fortunate in securing specimens of kidney from trout caught in the Sacramento River, 15 miles south of Redding, Calif. These kidney specimens contained cysts of a trematode which, when fed to a susceptible dog, produced a pathological syndrome typical of salmon poisoning. This is the first time that this parasitic disease has been reported in California streams south of the Eel River, in Humboldt County.

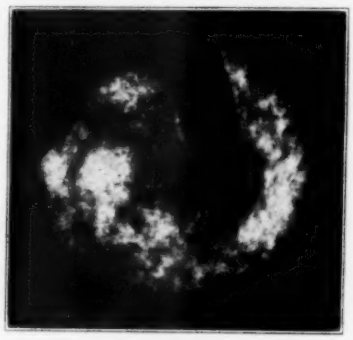


Fig. 1. Photomicrograph of encysted fluke.

The Sacramento River, therefore, seems to be a new habitat of the snail host of this parasite, since this fluke, *Nanophyetus salmincola*, Chapin, passes a part of its life cycle in the body of the snail, *Goniobasis plicifera*, var. silcula. As yet, the river banks have not been examined for the presence of the snail.

If the habitat of the parasite has extended southward, there is every reason to believe that small animal practitioners

*Received for publication March 6, 1939.

Anthrax in Farm-Raised Mink in Oregon*

By C. R. HOWARTH *and* L. SEGHETTI, *Corvallis, Ore.*

Anthrax occurs in practically all animals, domestic and wild. The herbivorous animals are most susceptible. Considerably less susceptible are the carnivores, such as the dog, cat and fox. Infection generally takes place through the ingestion of food or water containing anthrax spores. From an economic and public health standpoint, the disease is of great importance, because of the severity of the infection, the heavy losses that occur and the resistance of the organism to adverse exposure.

Sudden losses on fur farms are often attributed to improper feeding conditions. Frequently, domestic animals that are affected with some disease or that have died of an unknown cause are used for food. In a review of the literature, no report was found pertaining to the occurrence of this disease in mink. Hence, it seemed advisable to report this sporadic outbreak in Oregon.

HISTORY AND PATHOLOGY

Four pelted mink carcasses were brought to the laboratory from an area in which anthrax has never been reported. Very little of the previous history of the mink could be obtained. The owner stated that he was feeding a diet of fresh-frozen salmon heads and calf meat. The history of the calf carcasses used for food was incomplete and it has been impossible to trace the origin.

*Published as technical paper No. 307, with the approval of the Director of the Oregon Agricultural Experiment Station. Received for publication, February 20, 1939.

The carcasses were in good flesh and the animals were apparently in good health up to the time of death. Necropsy revealed that the viscera were congested. The liver and spleen were enlarged, dark red, and very friable. Lesions elsewhere were insignificant.

BACTERIOLOGICAL EXAMINATION

The heart blood, liver and spleen showed apparently pure cultures of large Gram-positive rods in large numbers. Anaerobic cultures from the spleen were negative but aerobic cultures produced luxuriant growths. Small amounts of inoculum from the cultures proved lethal to guinea pigs and the postmortem lesions resembled anthrax.

Morphologically, the organism is a relatively large Gram-positive bacillus, with truncate ends, and it grows in tangles of long threads, forming long chains of organisms. Spores were formed freely on a suitable medium and occurred either centrally within the bacillus or as free spores. Stained smears from the animal body did not show the presence of spores and the organisms occurred singly or in short chains. Encapsulated organisms could be demonstrated from cultures in serum broth or from smears obtained from animal tissues. In hanging drop preparations, no motility was observed.

The growth of the organism on serum-agar slants was spreading, flat, dull, and gray, with curled edges, and of membranous consistency. In serum broth there was a moderate floccular growth, with no turbidity. The growth in gelatin stabs was filiform and the medium was slowly liquefied. Litmus milk was slowly acidified and coagulated. Saccharose, maltose and dextrose were fermented with the production of acid but no gas was given off.

Due to the peculiarity of this case and to satisfy ourselves that the organism we had recovered from mink was *Bacillus an-*

(*Continued from page* 432.)

should suspect this infestation in dogs with clinical symptoms similar to distemper which do not respond to distemper treatment. This is an especially important consideration for practitioners in the San Francisco Bay region and those localities tributary to the Sacramento River watershed.

Paratyphoid and Trichomonas Infection in Pigeons*

By W. E. NIEMEYER, *Los Angeles, Calif.*

A disease that broke out in a pigeon loft in southern California, in 1938, was diagnosed as concurrent paratyphoid and trichomonas infections. There were several interesting aspects which seemed worthy of reporting, since there is comparatively little in the English literature dealing with such cases and since this is the first time that we have encountered a trichomonas infection in pigeons in southern California. It also is becoming evident that paratyphoid infections are growing in significance as a public health problem.

History and symptoms: The owner started to raise pigeons in September, 1937. Twenty-four pairs of breeding birds were purchased from Boston, Mass., and 44 pairs from Venice, Calif. In connection with the latter loft there was a history of disease, with but a few squabs being raised. At the time of delivery, a few of these birds had cankers in the mouth. Nothing could be learned of the state of health of the Massachusetts birds previous to their arrival in California.

In January, 1938, the first squabs developed a discharge from the nostrils and eyes, accompanied with a diarrhea. During the ensuing seven months, approximately 30 per cent of all the birds, both adult and young, developed similar symptoms and 60 squabs died. In the meantime, the owner, believing that his birds were affected with colds, had gone to considerable expense in inclosing one side of the pigeon house and using various remedies.

Autopsy findings: During August, 1938, when the outbreak was first called to our attention, autopsy examinations were made on 14 pigeons. In almost all instances, there was a mucopurulent discharge from the nostrils, slimy exudate in the oral cavity and often caseated material in the esophagus. The livers of four squabs were swollen and congested; on the surface of three there were small, gray foci. Three had enlarged spleens and the pericardial sac of one was distended with mucopurulent exudate. In four other birds small necrotic areas were found in the lungs, and in three the abdominal air sacs were distended with desiccated purulent exudate.

Microscopic examination of fresh material from the oral cavity, sinuses, trachea, esophagus and crop revealed large numbers of flagellates. These were identified by Dr. Ethel McNeil of the University of California as *Trichomonas columbae*. In the small intestine and bursa of Fabricius of a few birds, other protozoan flagellates were found and were later identified by her as a species of Hexamita. In one squab, approximately three weeks old, trichomonads were recovered from the liver and spleen.

Bacteriological studies: Cultures from the heart blood, liver and spleen of all birds were made routinely. From the livers

*Received for publication, March 4, 1939.

(*Continued from page* 433.)

thracis, we inoculated not only guinea pigs to reproduce the disease but also a sheep.

One sheep was inoculated subcutaneously with 1 cc. of a saline suspension of a 24-hour culture. The symptoms induced and the postmortem lesions were those of anthrax. Gram-positive rods were recovered in pure culture from the heart blood, spleen, and peritoneum.

SUMMARY

A Gram-positive bacillus, morphologically and culturally identical with *Bacillus anthracis*, was isolated from mink. Transmission experiments in guinea pigs and a sheep produced symptoms and lesions typical of anthrax infection.

of three a Gram-negative, motile rod was recovered that developed small, dense, gray colonies on meat-extract agar slants. It produced acid and gas in glucose and mannite media but lactose, sucrose and maltose were not attacked. This organism was identified by Edwards, of the University of Kentucky, as *Salmonella typhi-murium*—type IV variant. This same organism has been described by Jungherr and Wilcox,[1] in a paratyphoid outbreak in pigeons in New England, and by Hoffman and Edwards,[2] from California.

Serological tests: Serological tests were conducted on one pen of five adult breeding birds whose young had a history of poor livability. These five birds failed to agglutinate an autogenous antigen prepared from a dead squab. Tests conducted with a polyvalent antigen prepared from three other strains of *S. typhi-murium* were also negative. This does not eliminate the possibility of their having been infected, since other workers[1] have found that the agglutination test is not a reliable means of detecting carriers of this organism.

Transmission experiments: Krijgsman[3] reported that he was unable to transmit the infection, but Waller[4] succeeded by feeding the organisms to susceptible pigeons. Callender and Simmons[5] transmitted the infection to parrakeets, doves and chickens; in the latter birds the infection was transitory.

On September 27, 1938, a six-week-old Barred Rock pullet was inoculated *per os* with 2 cc. of mucus obtained from the oral cavity of an affected squab. Ten days later, material from the mouth of this pullet revealed many trichomonads, but 25 days following inoculation no flagellates could be found. Two more diseased squabs were received in the laboratory on October 6, both of which, upon autopsy, presented lesions similar to those previously described. Material from the oral cavity, sinuses, and crop was inoculated directly into the oral cavity of two apparently normal eight-week-old pigeons from another source. Both inoculated birds remained apparently healthy until they were destroyed eight weeks later. *T. columbae* were recovered from the upper respiratory and digestive tracts. In one pigeon small, gray, necrotic areas were seen in the liver, but we were unable to demonstrate the presence of flagellates in these lesions.

Four three-month-old pigeons were inoculated in the manner previously described with material obtained from sick or dead specimens. Twenty-five days later, one of the inoculated birds died, one was emaciated and the other two were apparently normal. Examination of the dead squab showed an enlarged, friable liver and spleen with yellowish colored exudate over the surfaces. The heart muscle was flaccid and the pericardial sac was distended with a serous fluid. The abdominal air sacs were thickened and their cavities filled with purulent exudate. There was a grayish colored, tenacious exudate and canker in the oral cavity and crop, and the liver showed a few small, gray areas on the surface. Examination of the emaciated bird revealed similar lesions, varying only in the degree of severity. In the remaining two birds which were clinically normal. *T. columbae* could not be demonstrated, although one showed suggestive lesions in the liver. Bacteriological examinations were carried out on all of the inoculated birds, but in no instance were we able to recover Salmonella.

REFERENCES

[1]Jungherr, E., and Wilcox, K. S.: *Salmonella aertrycke* variant as an etiologic agent of paratyphoid in pigeons. Jour. Inf. Dis., lv (1934), pp. 390-401.

[2]Hoffman, H. A., and Edwards, P. R.: The spontaneous transmission of IV-variants of *Salmonella aertrycke* from pigeons to rabbits. Amer. Jour. Hyg., xxvi (1937), pp. 135-137.

[3]Krijgsman, B. J.: Handbuch der Geflugel-Krankheiten und der Geflugelzucht. Trans., J. J. Posell. (Ferdinand Enke, Stuttgart, 1929), pp. 360-363.

[4]Waller, E. F.: A preliminary report on trichomoniasis of pigeons. Jour. A. V. M. A., lxxxiv (1934), n. s. 37 (4), pp. 596-602.

[5]Callender, G. R., and Simmons, J. S.: Trichomoniasis (*T. columbae*) in Java sparrow, Tovi parrakeet and Verraux's dove. Amer. Jour. Trop. Med., xvii (1937), 4.

Botulism in Foxes*

By NORMAN J. PYLE *and* RICHARD M. BROWN, *Pearl River, N. Y.*

Subsequent to the occurrence of a sudden and heavy mortality among silver foxes on a ranch in the East and the discovery that approximately 20 per cent of the sealed cans containing a certain brand of fish, which had been fed these foxes, were either swollen or had burst open at a seam, there were delivered at these laboratories for examination the carcasses of nine foxes, as well as six swollen 6-lb. cans of the fish, two apparently normal cans of the same product and three pieces of frozen horse meat from a lot which was part of the ration fed the foxes.

Results of Examination of Fox Carcasses

With but little variance in lesions, all carcasses showed the following autopsy picture: There was no discharge from the eyes or nasal passages, but some slight evidence of a conjunctivitis. The pleura and pericardium were normal. The lungs were either normal or slightly congested. The spleen was normal in size but somewhat degenerated in some of the carcasses. There was an acute nephritis in all cases. The gastrointestinal mucosa, in all carcasses, presented various degrees of congestion which in most cases was severe, with an oozing of blood into the lumen of the intestine. In two of the specimens, the mucosa of the intestine was also severely catarrhal with complete destruction. The bladder was distended in all of the carcasses. No parasites were found.

In all nine foxes, careful bacteriological examinations (aerobic cultures) were made of the heart blood, lungs, liver, spleen and kidneys. These were negative, except for one case in which the colon organism was found in the lung tissue. In three of the carcasses, aerobic cultures of the tonsils also were made. One tonsillar examination disclosed the presence of *Staphylococcus albus* and the colon organism; a second, *S. albus* alone; the third, negative results.

Both the pathological and bacteriological findings in these cases strongly suggested that the mortality might have been due to an acute toxemia.

Results of Physical and Bacteriological Examinations of the Canned Fish

When the six swollen cans of fish were opened it was quite apparent that there had been considerable gas formation. The contents emitted various degrees of putrid odors and the fish was darker and more moist than that in the two normal cans opened and examined at the same time.

These results led to the suspicion that the spoiled fish contained the gas-forming botulinus organism which produces a very active and invariably fatal toxin for some animals, particularly for foxes.

As further evidence that a suspicion of botulinus infection was justified, Jones[1] states:

> Food infected with organisms of the Salmonella group usually appears quite normal; there need be no great change in color and no detectable change in taste or smell. But in the case of botulism, the preserved foods are frequently noticeably spoiled, the cans are usually "blown," the food shows evidence of decomposition in varying degrees, and it has a more or less rancid odor.

According to Krampert,[2] the source of botulinus infection in two human patients was traced to spoiled canned fish which both had eaten. In the first case, a girl of six years, 20 cc. of specific antitoxin effected a cure in one week. In the other case, a man of 36 years, 2,000 units of the bivalent antitoxin caused a sharp improvement in 24 hours.

Aerobic cultures of the contents of the swollen cans revealed the presence of the colon organism and *S. albus* in all cases. No organisms were isolated from the contents of the apparently normal cans.

*Received for publication, November 16, 1938.

These results would indicate that the particular lot of fish, of which the six swollen cans were a part, was not properly sterilized at the canning factory. Otherwise, the unbroken, bulging cans would not have contained these organisms. As none of these cans were "leakers," there was no possibility of external contamination.

At the same time that this work was done and in order to determine the possible presence of *Clostridium botulinum* in the contents of the bulged cans, five grams from each of such cans was placed in each of several tubes of anaerobic meat media. These tubes, with their contents, were then heated at 85° C. for 30 minutes for the purpose of destroying all aerobic, contaminating organisms. Then they were incubated at 37.5° C. for two weeks, at the end of which time no Gram-positive, sporulating, rod-shaped organisms were found in the medium.

Thus we were unable to demonstrate the botulinus organism in the contents of the bulged cans of fish. Such results, however, when one deals with this organism, are not unusual. While the bacteriological examination of the spoiled fish was negative in this single trial, such a result did not eliminate the possible presence of botulinus toxin in the same material. Accordingly, the following experiment was undertaken.

Results of Animal Inoculations with Spoiled Fish and Stomach Contents of Fox Carcasses

About 500 grams of the contents of each of the swollen cans and the normal cans were placed in separate two-liter flasks and enough sterile physiologic saline solution added to make a mush consistency. The contents of these flasks were then agitated and centrifuged sufficiently to give about 200 cc. of supernatant fluid in each. The two separate supernatants were next filtered through a Seitz filter.

During the autopsy of the fox carcasses, the stomach contents were removed, pooled and centrifuged and the supernatant fluid was passed through a Seitz filter.

In table I, the filtered supernatant from the spoiled fish is designated "spoiled filtrate" and that from the normal fish, "unspoiled filtrate." The end product of the stomach contents is designated "stomach filtrate." All injections were made subcutaneously into guinea pigs.

Interpretation of results: It will be seen that doses of 5 cc. and 3 cc. of the spoiled fish filtrate killed guinea pigs in 48 hours. Since all filtrates were proved to be bacteriologically sterile before being injected, it is apparent that the lethal factor was a toxin. Furthermore, when 3 cc. of this filtrate was mixed with 2 cc. of botulinus antitoxin (types A and B), the lethal dose of toxin was neutralized. There is an indication, therefore, that the contents of the swollen cans of fish contained the botulinus toxin.

No toxin was demonstrated in the unspoiled filtrate, *viz.*, the contents of the normal cans. Likewise, toxin was not found in the stomach contents of the fox carcasses. With regard to the latter result, it is quite likely that all of the ingested toxin had passed from the stomach into the intestines and, from there, was absorbed into the general circulation.

In making the tests shown in table I, all guinea pigs were injected subcutaneously with the material suspected of containing botulinus toxin, because it is only through such a method of administration that it is possible to demonstrate a weak botulinus toxin. Feeding experiments will fail in the test animal, unless the toxin is highly potent. In this connection, it should be borne in mind that the fox is very susceptible even to small amounts of this toxin when it is ingested with the feed.

Results of Attempts to Determine the Titre of the Toxin in the Spoiled Fish and to Confirm Its Identity

Since it is apparent that botulinus toxin was present in the spoiled fish, further tests were made in an attempt to determine roughly the minimum lethal dose. Increasing doses of the spoiled filtrate were used for this purpose.

TABLE I—*The presence and identiity of a toxin in spoiled fish, unspoiled fish and stomach contents.*

G. P.	DOSE (CC.)	MATERIAL INJECTED	RESULTS
1	5	Spoiled filtrate	Death—48 hrs.
2	3	Spoiled filtrate	Death—48 hrs.
3	3	Spoiled filtrate plus 2 cc. antitoxin*	Lived
4	5	Unspoiled filtrate	Lived
5	5	Stomach filtrate	Lived
6	3	Stomach filtrate	Lived
7	3	Stomach filtrate plus 2 cc. antitoxin*	Lived
8	5	Antitoxin* (control)	Lived

*Botulinus antitoxin, bivalent A and B.

TABLE II—*The titre and identity of toxin in spoiled fish.*

G. P.	DOSE (CC.)	MATERIAL INJECTED	RESULTS
9	0.5	Spoiled filtrate	Lived
10	1.0	Spoiled filtrate	Death—7 days
11	2.0	Spoiled filtrate	Death—3 days
12	3.0	Spoiled filtrate	Death—2 days
13	4.0	Spoiled filtrate	Death—2 days
14	5.0	Spoiled filtrate	Death—2 days
15	0.5	Spoiled filtrate plus 2 cc. botulinus antitoxin, type A	Lived
16	2.0	Spoiled filtrate plus 2 cc. botulinus antitoxin, type A	Lived
17	3.0	Spoiled filtrate plus 2 cc. botulinus antitoxin, type A	Death—3 days
18	0.5	Spoiled filtrate plus 2 cc. botulinus antitoxin, type B	Lived
19	2.0	Spoiled filtrate plus 2 cc. botulinus antitoxin, type B	Death—2 days
20	3.0	Spoiled filtrate plus 2 cc. botulinus antitoxin, type B	Death—2 days

At the same time, in order to confirm the identity of the toxin, various mixtures of the spoiled filtrate and botulinus antitoxin, types A and B, were administered subcutaneously into guinea pigs.

Interpretation of results: The results shown in table II indicate that the smallest lethal doses of the toxin were 1 cc., which killed a guinea pig in seven days, and 2 cc., which caused the death of a pig in three days.

Apparently, 2 cc. of botulinus antitoxin, type A, was sufficient to neutralize 2 cc. of the toxin, but not 3 cc. (pig 17). However, guinea pig 17, which died in three days, did not show the characteristic flaccidness typical of clinical botulinus intoxication. All other test animals, in both table I and II, in which death was attributed to the toxin, showed this symptom.

It will be seen in table II that botulinus antitoxin, type B, failed to neutralize doses of 2 cc. and 3 cc. of the toxin.

These results further indicate that the lethal substance in the spoiled fish was botulinus toxin and that apparently it was type A.

RESULTS OF FEEDING EXPERIMENT

Although it is definitely known that a weak botulinus toxin will not kill guinea pigs when it is administered orally, it was decided to attempt a feeding experiment. In this trial, the spoiled filtrate was administered orally by means of a small dropper pipette in the doses listed in table III.

TABLE III—*Results of feeding guinea pigs with spoiled filtrate.*

G. P.	SPOILED FILTRATE (CC.)	RESULTS
21	2	Lived
22	4	Lived
23	6	Lived
24	8	Death—12 days

Interpretation of results: Although the number of experimental subjects is small, there is some indication that relatively small doses of the spoiled filtrate, when administered orally, were not lethal for guinea pigs. Larger doses apparently were fatal. In this case, 8 cc. killed a guinea pig in twelve days with characteristic symptoms of botulinus infection.

Summary and Conclusions

Immediately following an outbreak of disease with heavy mortality among silver foxes on a ranch in the East, a number of fox carcasses and representative samples of suspected spoiled fish were examined in an effort to determine the cause of death. Apparently normal fish and frozen horse meat, which were part of the ration, also were examined at the same time.

An autopsy and bacteriological examination of nine fox carcasses indicated, in all cases, that death had been due to an acute toxemia. No parasites or pathogenic organisms were found in the animals. While the colon organism, *Staphylococcus albus* and *Staphylococcus aureus* were isolated from the frozen meat, there was no indication that this component of the ration was a cause of the mortality.

The examination of the several swollen cans of fish revealed that the contents were darker and more moist than were the contents of the cans which were normal. The apparently spoiled fish also gave forth a rancid or putrid odor. Aerobic cultures of the contents of the swollen cans revealed the colon organisms and *S. albus*. The contents of the normal cans were bacteriologically sterile. Anaerobic cultures of the contents of the swollen cans failed to reveal the presence of *Clostridium botulinum*. When one deals with this organism, however, such a result is not unusual.

Filtered extracts were made of the contents of the stomachs of the fox carcasses, the spoiled fish and the apparently normal fish. A toxic substance was found only in the spoiled fish. This killed guinea pigs, upon subcutaneous injection, in a minimum dose of 1 cc. in seven days. A dose of 2 cc. killed in three days, and doses of 3 cc., 4 cc. and 5 cc. killed the test animals in two days. All guinea pigs showed characteristic symptoms of botulinus intoxication.

Since 2 cc. of a bivalent (types A and B) botulinus antitoxin neutralized 3 cc. of the toxin, it was concluded that the lethal substance was botulinus toxin. Further study of the toxin demonstrated that it was neutralized with type A botulinus antitoxin. Since this was not accomplished with type B botulinus antitoxin, it was apparent that the spoiled fish contained type A toxin.

A superficial attempt to kill guinea pigs by feeding the toxin indicated that at least 8 cc. of the spoiled filtrate material was required. This was to be expected, as guinea pigs are relatively resistant to botulinus toxin administered orally.

References

[1]Jones: Food poisoning—its epidemiology and bacteriology. Brit. Med. Jour., 3933 (1937), pp. 106-110.

[2]Krampert: Report of two cases of botulism. Neb. State Med. Jour., xxii (1937), p. 189.

Problems of Fur Farms Studied

Fur farms in the United States, following a rapid development in the past 15 years, now produce about 300,000 silver fox and 200,000 mink pelts annually, the U. S. Biological Survey points out, yet many fundamental questions on handling these wild animals have never been answered well enough to prevent heavy losses to fur farmers.

Nutrition, one of the fundamental problems, is now being studied in a coöperative five-year project recently inaugurated by the Survey and the Laboratory of Animal Nutrition at Cornell University, Ithaca, N. Y. Animals for this study have been furnished by the U. S. Fur Animal Experiment Station, maintained by the Survey, near Saratoga Springs, N. Y.

Applied veterinary science is no doubt the greatest salvage service of this civilization.

Fibrosarcoma*

By R. L. BOOTH, *Middleburg, Va.*, and J. RAYMOND CURREY, *Washington, D. C.*

The subject, a male Pointer dog, three years of age, was examined on March 30, 1938. The right hind leg was a bit lame and there was a slight fullness of the posterior right sacroiliac region and right perineum. No history was available.

March 31: The temperature was slightly elevated and the appetite poor. The swelling had enlarged, and digital examination of the interior of the pelvis revealed a firm mass in the right half of the cavity. This apparently did not inconvenience the patient and it did not fluctuate. The mass was in close proximity to the prostate gland, which seemed to be normal in size and consistency.

April 1: Temperature still elevated; increased swelling in perineum; skin to side of anus slightly macerated, with oozing of reddish, serosanguineous fluid. A tentative diagnosis—pelvic cellulitis, possibly of streptococcic origin—was made. Treatment consisted of 10 cc. of Prontosil injected into the gluteal muscles and sulfanilamide in 10-grain doses every four hours. Two cc. of Omnadin also was administered, the pelvis bathed and a heat pad applied.

April 2: Condition about same; pelvic mass about same size; sulfanilamide, baths and heat pad continued.

April 3: The feces were bloody, but examination of the rectum, digitally with the aid of a speculum, did not reveal the pointing of the pelvic mass into the rectum. Omnadin, 2 cc., was repeated, baths and heat pad continued, and sodium bicarbonate in 10-grain doses given every four hours.

April 4: Temperature higher; skin tonus very poor; patient very depressed; appetite gone, with death apparently impending; same treatment continued; temperature low normal.

April 5: Condition greatly improved; temperature normal. Colonic irrigation revealed a considerable amount of bloody mucus in the bowel. Sulfanilamide dosage was cut down and Omnadin treatment repeated.

April 6: Continued improvement; better appetite. The pelvic mass seemed to recede but mucosanguineous stools continued.

April 7-25: Steady improvement; pelvic mass receded. The animal was gaining in flesh but the tail movement was still impeded. Bloody stools continued intermittently.

April 29: Hookworm diagnosis made; pelvic mass apparently gone.

April 30: Dog "wormed" with five Caprokol pills; thereafter, stools cleared up.

May 14: Subject "wormed" again, in same manner.

August 15: Swelling appeared in paralumbar region; quite solid, but flat and diffuse; antiphlogistine applied daily for several days.

August 28: Swelling larger and softer; lanced, small amount of serosanguineous fluid escaping; poultice continued.

September 1: Parasite examination, negative.

September 3: The animal was x-rayed at the Winchester (Va.) Memorial Hospital. The plates revealed a poorly defined mass in the abdominal wall in the paralumbar region, but there was no definite evidence of fluid accumulation.

September 8: Swelling lanced again; poultice continued. (Case was turned over to the junior author on this date.)

October 3: Subject in very good condition; bright and active; no lameness or soreness in involved region.

The presence of a well defined, circumscribed mass, coupled with past history of recurrent fluctuation, prompted a radical surgical procedure as a last hope to giving the animal ultimate relief.

Following incision of the skin, exploration revealed the presence of numerous

*Received for publication, March 10, 1939.

fistuli radiating from the center of the mass, the two larger of which lead directly dorsoposteriorly to the lumbar vertebrae and the others to the wall of the pelvis. Dr. Booth had been able to detect these by digital examination. The color, character and consistency of the incised tissue suggested the presence of a malignancy and, from then on, exploration was continued with no thought of repair or recovery. The mass involved considerably more tissue than could be determined prior to surgical intervention; it was definitely established even adjacent to the peritoneum.

Euthanasia was performed and the involved section removed for pathological classification.

Postmortem: Gross examination provided very little additional information.

This is the third case of a similar nature that the operator (Currey) has handled. Strangely, both of the other cases were Pointers (bitches), with the growth involving the right perineal region. In each instance growth recurred, after surgical excision, within two months. These previous cases concur with Dr. Hunter's prognosis.

Infectious Equine Encephalomyelitis: Mid-Winter Case*

By O. L. OSTEEN, *Washington, D. C.*
Pathological Division, U. S. Bureau of Animal Industry

The available epizoötiological evidence points to infectious equine encephalomyelitis as a strictly seasonal disease. Prior to the case herein reported, the earliest cases have been reported to the U. S. Bureau of Animal Industry from Florida, in late May or early June. For the United States in general, however, July, August and September are the months during which the disease is most prevalent. It is altogether probable that a few authentic cases occur as early as June and as late as November, but these are exceptional.

During the first week of June 1937, Dr. R. L. Brinkman of Live Oak, Fla., encountered clinical cases of the disease in Florida and forwarded to the Pathological Division a specimen of brain taken from an animal which had exhibited typical symptoms. Although inoculation tests in guinea pigs failed to reveal the presence of virus, histopathological examination disclosed alterations in the brain characteristic of the virus disease. During the summer and fall of 1937, other specimens of brain tissue were received from Florida and, although virus was not recovered in any instance, pathological changes diagnostic of virus encephalomyelitis were found in most of the specimens examined.

During the 1938 outbreak, many brain specimens were received from Florida and, again, histopathological changes typical of those of the virus disease were encountered, and in one specimen virus which proved to be true eastern-type equine encephalomyelitis virus was recovered.

On January 13 of this year (1939) the Bureau received from Dr. E. F. Thomas, of Ocala, Marion County, Fla., a specimen of horse-brain tissue taken from an animal which had died of a disease producing clinical symptoms similar to those of infectious equine encephalomyelitis.

Because of an oversight, this specimen was placed in refrigeration in its original container and examination was not begun until January 31. At this time, a saline suspension of the brain tissue was prepared and inoculated intracranially into four normal guinea pigs. Part of the emulsion was filtered and the filtrate inoculated into eleven-day chick embryos. Sections were prepared and histopathological examination revealed changes characteristic of the virus of encephalomyelitis. No virus was recov-

*Received for publication, March 15, 1939.

ered from the embryos inoculated with the filtrate.

Of the four guinea pigs inoculated, three remained normal. The other one appeared to be abnormal on the fourth day following inoculation. Partial paralysis was evident on the fifth day and, on the sixth day, the animal was completely paralyzed and exhibited symptoms typical of those produced by the virus of encephalomyelitis. At this time, the animal was chloroformed and the brain removed, cultures from which remained free from bacterial growth. Saline emulsion of this brain was inoculated intracranially into three additional normal guinea pigs.

Sections of the brain also were examined and found to show characteristic changes of encephalomyelitis. Of the three guinea pigs inoculated, two were dead on the third day, the third being in a state of complete paralysis. Cultures from each of the brains of these three animals also remained free from bacterial growth. With portions of these three brains, pooled, a typing test was conducted, which proved the infectious agent to be the eastern-type virus of infectious equine encephalomyelitis.

In correspondence received from Dr. Thomas on January 28, 1939, in which he related typical symptoms of encephalomyelitis in the case referred to above, he stated that none of the animals on this farm had been vaccinated, whereas other animals in his locality had been vaccinated and in these the disease had not appeared. He further stated that the weather had been very mild during the winter, a factor which he believed contributed to the presence of the disease at this time of the year.

Dr. F. C. Bishop, of the U. S. Bureau of Entomology, informs the writer that it is not unusual to find mosquitoes active as far north as Ocala, Fla., in the month of January. Moreover, the weather reports show that the Florida temperatures for the week ended January 10, 1939, were eight or nine degrees above normal and there is no doubt that there was considerable mosquito activity at that time.

(Discussion of Dr. Reichel's paper, page 418.)

DR. J. D. RAY: I would like to ask Dr. Reichel if, in his experience, it has been practical to desensitize animals that were about to be treated with biologics. I have sheep in mind particularly. Just about every so often we have a flock of sheep that will react very strongly to the introduction of a biologic. It is just the occasional flock that will have a tendency to react. We might vaccinate 50 flocks and never have any trouble.

DR. REICHEL: I can readily answer that I do not think it would be practical, but I do think it is possible. If nothing else be done, the method of injection should be carefully considered. You might hesitate to make an intravenous injection under such circumstances, whereas you might make an intramuscular injection. In a sensitized animal, you have to grade your dose very carefully to start with, take considerable time and make repeated injections, before you have the dose that you had planned to give the animal, and that is not practical.

DR. T. H. FERGUSON: In a case of a few cases of anaphylactic shock, how would you handle them clinically?

DR. REICHEL: You have to take into consideration symptoms, inasmuch as the symptoms have to do with the respiratory system and possibly the heart. The procedure is to stimulate both, and adrenalin in large doses tends to make the animal recover much more quickly.

DR. FERGUSON: That had been my experience—that a dose of adrenalin chloride would probably bring them out of it.

DR. REICHEL: The surprising thing about the reaction is the severity of the symptoms, and that there are but few deaths. I did not think there were quite so many deaths before I started on a western journey about five weeks ago. I heard then of a number of deaths, following the injection of heated anti-hog cholera serum in young pigs, from typical anaphylactic reactions, even when injected for the first time. These cases are not negligible.

DR. H. F. FLEMING: I would like to ask Dr. Reichel if he has had any experience along this line: I have a farm on which I have experienced difficulty in treating pigs about three weeks of age. Every once in a while I find a litter that is particularly susceptible to anaphylactic reaction. We tried to inject one or two pigs in a litter, at first. In that way I locate my susceptible litter. On one occasion, every pig in the litter died before I tried that system. Recently I have tried administering 0.5 cc of adrenalin mixed right with my serum in administering the serum. Since I have done that, I have had no bad results.

DR. REICHEL: Frankly, I think that is a very fine, practical procedure. The simultaneous use of a very small dose of adrenalin has been found to reduce anaphylactic possibilities. I would like to suggest that you make your injections intramuscularly, too, rather than into the axilla.

LEGAL OPINIONS

By JOSEPH M. KOTZ, *Attorney for the Association*

Death of Sheep from Drenching

An interesting case on this question is that of *E*— vs. *W*—, 237 N. W. 558. The defendants, veterinarians practicing under a co-partnership, were charged by the plaintiffs with negligence in compounding and administering a remedy for intestinal worms in sheep.

After purchasing approximately 90 sheep, the plaintiffs requested the veterinarians to examine them. The doctors reported, after examination, that the animals were in good condition but advised that a treatment be given to pave the way for sound growth. The owners agreed and, on the same morning that the sheep were treated, some were found dead and others were lying on the ground, with a greenish foam issuing from the mouth. During the afternoon and night of the same day, 40 of the animals had died and, within a few days, 19 more had succumbed.

The case was tried and a verdict rendered in favor of the owners.

A veterinarian was called in by the plaintiffs as a witness. He was asked to testify on the basis of an assumed state of facts. He contended that the deaths were caused by faulty administration of the medicine, that is, the medicine had gone into the lungs.

The defendants claimed, however, that there was no correlation between the alleged negligence and the loss of the animals, that the deaths were not evidence of neglect on their part, and that there was no reliable way to determine the responsibility in the case.

There was uncontradicted evidence that the medicine was properly compounded and, therefore, the case rested upon proving negligence in administering the remedy. Experts testified that, when liquid identical with that used in treating the sheep is not properly administered, it passes into the lungs and causes strangulation and almost certain death. This testimony was substantiated by reference to a treatise on the diseases of sheep, written by recognized authorities and issued by the U. S. department of Agriculture.

From the testimony given, it was concluded that the manner in which the treatment was administered is usually fatal. The court held that the mere fact that injury resulted from the use of this remedy in the way it was intended to be used was not sufficient proof of negligence in its manufacture. Judgment was affirmed.

Deaths Following Hog-Cholera Vaccination

In the case of *M*— vs. *X. Co.*, 195 N. W. 835, the defendant was a manufacturer of hog-cholera virus. He was accused of negligence in preparing the product used in treating the plaintiff's hogs.

As veterinarians know, hog cholera in its incipient stages is difficult to detect. A high death rate following preventive treatment for hog cholera, however, indicates either that the disease was present in its incipient stage at the time of treatment or that a defective remedy was used. In the words of the court: "It is pretty well established that, in its incipient stages, hog cholera can not be detected and, when it has advanced to the stage where the symptoms are discernible, it is too late for most hogs to yield to the serum. This is true of the disease whether resulting from the usual contagion or caused by the injection of the virus."

In the case of *B* — vs. *X. Co.*, 200 N. W. 601, the plaintiff charged that the company's serum and virus, publicly stated as a proper compound for immunization against hog cholera, did not fill the purpose for which it was manufactured and sold. It was further charged that the company was negligent in the manufacture and sale of this product, since the compound failed to conform to the laws of the state. Suit was brought on the grounds that, had the product conformed to legal requirements, the plaintiff would not have been damaged.

A veterinarian had vaccinated the hogs, and evidence showed that the serum had been administered properly and the animals given proper care after the treatment. It was pointed out that, about 1½ months afterward, one of the hogs had become sick and died in two or three days and, within another month, 30 more were dead and others in the herd were affected.

The question involved was whether or not the manufacturer of a serum, intended and claimed as a preventive for a certain kind of loss, is liable for damages caused by the failure of his product to prevent the loss.

The court held that, in the sale of vaccine or serum for the inoculation of animals against some specific disease, the seller or manufacturer is not a guarantor or warrantor of the efficacy of the remedy and can not be held liable for its failure in that regard, except upon proof of negligence either in the manufacture or putting up of the product.

In the case of *R*— vs. *X. Co.*, 236 Fed. 677, it was held that, if damages were to be recovered, on the grounds of negligence in manufacture or breach of warranty, the plaintiff must establish the fact that the vaccine was infective at the time of purchase. If he is unable to establish this, he can not recover.

Likewise, in the case of *H*— vs. *X. Co.*, 162 N. W. 620, the court held that the loss of a large number of hogs, following inoculation with a given serum, does not in itself prove negligence on the part of the manufacturer.

It has been held, however, that where a company manufacturing serum to inoculate hogs for cholera undertakes, through its employés and agents, to perform the act of inoculation, and where this act results in the death of such hogs, due to the careless manner of administration, the manufacturer may be held liable (*S*— vs. *X. Co.*, 204 Pac. 988).

Quoting from the case of *X. Co.* vs. *C*—, 88 S. W. 873:

> Where the seller of vaccine virus which bears a label indicating it to be of a certain kind, and to be used only upon certain animals (cattle), represents to the buyer that it is the same as a virus of a different name ordered by the latter and that it can be used for the purpose for which the virus was ordered (to vaccinate horses), the seller is liable to the buyer for the damage resulting to the latter by the death of several of his horses which he inoculated with this virus in reliance upon the seller's statements.

"And Sudden Death"

Three million persons are seriously ill in this country today.

One thousand eight hundred different diseases are now known to exist.

Thirty thousand people at home today will enter a hospital tomorrow.

Automobile fatalities have increased 500 per cent during the past 20 years.

Automobile accidents caused about 1,000,000 deaths and injuries in a year.

Accidents to men are 960 times more frequent than fire to their property.

Ninety-nine people are disabled from sickness or accident to every one that dies.

One hundred thousand persons are accidentally killed in the United States each year.

Four million five hundred persons are disabled annually as a result of injuries in the home.

CURRENT LITERATURE

ABSTRACTS

EFFECT OF SULFANILAMIDE ON PNEUMOCOCCAL INFECTIONS IN MICE. James M. Ruegsegger and Morton Hamberger. Jour. Inf. Dis., lxix (1939), p. 18.

Sulfanilamide administered parenterally tended to prolong the lives of mice inoculated with virulent strains of types I, II, III, and VIII pneumococci. No protective action was apparent in this series of experiments. Studies were not made on the exudates to attempt to learn the mode of action of the drug. Sulfanilamide has very little toxicity for the white mouse, except in large doses.

THE EFFECT OF SULFANILAMIDE THERAPY UPON BOVINE MASTITIS AS INDICATED BY LABORATORY TESTS. W. G. Hoge, W. V. Halversen and V. A. Cherrington. Jour. Inf. Dis., lxiv (1939), p. 27.

Blood-agar plate counts and Breed smears were made from the milk from each quarter of five experimental animals to determine the effect of the medication on the disease. Concentrations of sulfanilamide in the blood, milk and urine were determined, in order to establish any relationship between the amount present in the body fluids and the effect produced upon the condition of the udder.

The cows tested tolerated doses of 5 mg. per 100 pounds body weight per day (the dose often prescribed in human infections). In some cases this dose was doubled without producing serious symptoms of toxicity. Uniform blood and milk concentrations were well maintained by only two or three daily doses. The bacterial counts of the milk were greatly reduced during medication in all cases. In most cases the milk was temporarily rendered nearly or completely sterile by the drug.

Improvement in the clinical picture and in the milk was noted in the one case of acute mastitis tested. There was no permanent improvement in the udder as revealed by bacteriological examination of the milk or by the leukocyte count in any of the cases studied. There was a decrease in the number of leukocytes in the milk of treated cows. Sulfanilamide in litmus milk cultures of mastitis organisms markedly inhibits the action of the organisms on the milk. The effect of the drug *in vivo* appeared to be much greater than *in vitro*.

EIN FALL VON PATHOLOGISCHER VERKNÖCHERUNG IM BEREICH DER KEHLKOPFKNORPEL BEIM PFERD ALS BEITRAG ZUR KASUISTIK KOMPLIZIERTER KEHLKOPFERKRANKUNGEN. (Chronic laryngeal catarrh complicated with paralysis of the recurrent nerve and partial ossification of the thyroid cartilage in a ten-year-old army mount.) Siegmund. Zeit. f. Veterinärk., li (1939), p. 75.

Siegmund points out that in a case of chronic catarrh of the larynx failing to respond to treatment, one should also consider paralysis of the recurrent laryngeal nerve as a possible complication, even though roaring can not be diagnosed clinically and laryngoscopically. In the case under consideration, which was further complicated by a partial ossification of the base of the thyroid cartilage, it is plausible that the ossification already existed at the start of the lesions leading to the paralysis of the recurrent nerve; possibly, they contributed indirectly to the stenosis of the laryngeal pouch.

Undoubtedly, the unilateral paralysis of the recurrent laryngeal nerve and the pronounced ossification of the thyroid cartilage

In the case of *B*— vs. *X. Co.*, 200 N. W. 601, the plaintiff charged that the company's serum and virus, publicly stated as a proper compound for immunization against hog cholera, did not fill the purpose for which it was manufactured and sold. It was further charged that the company was negligent in the manufacture and sale of this product, since the compound failed to conform to the laws of the state. Suit was brought on the grounds that, had the product conformed to legal requirements, the plaintiff would not have been damaged.

A veterinarian had vaccinated the hogs, and evidence showed that the serum had been administered properly and the animals given proper care after the treatment. It was pointed out that, about 1½ months afterward, one of the hogs had become sick and died in two or three days and, within another month, 30 more were dead and others in the herd were affected.

The question involved was whether or not the manufacturer of a serum, intended and claimed as a preventive for a certain kind of loss, is liable for damages caused by the failure of his product to prevent the loss.

The court held that, in the sale of vaccine or serum for the inoculation of animals against some specific disease, the seller or manufacturer is not a guarantor or warrantor of the efficacy of the remedy and can not be held liable for its failure in that regard, except upon proof of negligence either in the manufacture or putting up of the product.

In the case of *R*— vs. *X. Co.*, 236 Fed. 677, it was held that, if damages were to be recovered, on the grounds of negligence in manufacture or breach of warranty, the plaintiff must establish the fact that the vaccine was infective at the time of purchase. If he is unable to establish this, he can not recover.

Likewise, in the case of *H*— vs. *X. Co.*, 162 N. W. 620, the court held that the loss of a large number of hogs, following inoculation with a given serum, does not in itself prove negligence on the part of the manufacturer.

It has been held, however, that where a company manufacturing serum to inoculate hogs for cholera undertakes, through its employés and agents, to perform the act of inoculation, and where this act results in the death of such hogs, due to the careless manner of administration, the manufacturer may be held liable (*S*— vs. *X. Co.*, 204 Pac. 988).

Quoting from the case of *X. Co.* vs. *C*—, 88 S. W. 873:

> Where the seller of vaccine virus which bears a label indicating it to be of a certain kind, and to be used only upon certain animals (cattle), represents to the buyer that it is the same as a virus of a different name ordered by the latter and that it can be used for the purpose for which the virus was ordered (to vaccinate horses), the seller is liable to the buyer for the damage resulting to the latter by the death of several of his horses which he inoculated with this virus in reliance upon the seller's statements.

"And Sudden Death"

Three million persons are seriously ill in this country today.

One thousand eight hundred different diseases are now known to exist.

Thirty thousand people at home today will enter a hospital tomorrow.

Automobile fatalities have increased 500 per cent during the past 20 years.

Automobile accidents caused about 1,000,000 deaths and injuries in a year.

Accidents to men are 960 times more frequent than fire to their property.

Ninety-nine people are disabled from sickness or accident to every one that dies.

One hundred thousand persons are accidentally killed in the United States each year.

Four million five hundred persons are disabled annually as a result of injuries in the home.

CURRENT LITERATURE

ABSTRACTS

EFFECT OF SULFANILAMIDE ON PNEUMOCOCCAL INFECTIONS IN MICE. James M. Ruegsegger and Morton Hamberger. Jour. Inf. Dis., lxix (1939), p. 18.

Sulfanilamide administered parenterally tended to prolong the lives of mice inoculated with virulent strains of types I, II, III, and VIII pneumococci. No protective action was apparent in this series of experiments. Studies were not made on the exudates to attempt to learn the mode of action of the drug. Sulfanilamide has very little toxicity for the white mouse, except in large doses.

THE EFFECT OF SULFANILAMIDE THERAPY UPON BOVINE MASTITIS AS INDICATED BY LABORATORY TESTS. W. G. Hoge, W. V. Halversen and V. A. Cherrington. Jour. Inf. Dis., lxiv (1939), p. 27.

Blood-agar plate counts and Breed smears were made from the milk from each quarter of five experimental animals to determine the effect of the medication on the disease. Concentrations of sulfanilamide in the blood, milk and urine were determined, in order to establish any relationship between the amount present in the body fluids and the effect produced upon the condition of the udder.

The cows tested tolerated doses of 5 mg. per 100 pounds body weight per day (the dose often prescribed in human infections). In some cases this dose was doubled without producing serious symptoms of toxicity. Uniform blood and milk concentrations were well maintained by only two or three daily doses. The bacterial counts of the milk were greatly reduced during medication in all cases. In most cases the milk was temporarily rendered nearly or completely sterile by the drug.

Improvement in the clinical picture and in the milk was noted in the one case of acute mastitis tested. There was no permanent improvement in the udder as revealed by bacteriological examination of the milk or by the leukocyte count in any of the cases studied. There was a decrease in the number of leukocytes in the milk of treated cows. Sulfanilamide in litmus milk cultures of mastitis organisms markedly inhibits the action of the organisms on the milk. The effect of the drug *in vivo* appeared to be much greater than *in vitro*.

EIN FALL VON PATHOLOGISCHER VERKNÖCHERUNG IM BEREICH DER KEHLKOPFKNORPEL BEIM PFERD ALS BEITRAG ZUR KASUISTIK KOMPLIZIERTER KEHLKOPFERKRANKUNGEN. (Chronic laryngeal catarrh complicated with paralysis of the recurrent nerve and partial ossification of the thyroid cartilage in a ten-year-old army mount.) Siegmund. Zeit. f. Veterinärk., li (1939), p. 75.

Siegmund points out that in a case of chronic catarrh of the larynx failing to respond to treatment, one should also consider paralysis of the recurrent laryngeal nerve as a possible complication, even though roaring can not be diagnosed clinically and laryngoscopically. In the case under consideration, which was further complicated by a partial ossification of the base of the thyroid cartilage, it is plausible that the ossification already existed at the start of the lesions leading to the paralysis of the recurrent nerve; possibly, they contributed indirectly to the stenosis of the laryngeal pouch.

Undoubtedly, the unilateral paralysis of the recurrent laryngeal nerve and the pronounced ossification of the thyroid cartilage

are causally connected with the chronic inflammation of the laryngeal mucosa. Each one of these disease conditions complicated the other in regard to existence and development. In the beginning the laryngeal catarrh was probably an ideopathic disease, but later was hastened and heightened by a venous congestion produced by the paralysis of the recurrent laryngeal nerve and the ossification of the base of the thyroid cartilage. These complications readily explain the uselessness of any treatment.

FLUORINE POISONING AND CALCIFICATION OF THE PERMANENT TEETH IN LAMBS. (Title translated.) H. Velu. Rev. de Méd. Vét., cxiv (1938), p. 805. Abstract from Compte Rendu de la Société de Biologie (1938) pp. 137, 854.

The author, in recent years, has studied the signs of fluorine poisoning of sheep in Morocco, where this intoxication is enzoötic in certain regions rich in phosphates. Fluorine is usually found associated with phosphorus.

Lambs experimentally intoxicated, divided into groups for study, were given feed and water supplemented with fluorine and calcium in the same proportions as these elements exist in the region. Water containing less than 7 mg. per liter was harmless but, when greater amounts were ingested, grave dental lesions supervened. No effect was observed in the deciduous teeth which erupted during the period of the feeding experiment. On the contrary, pronounced lesions developed in the permanent teeth whose dental follicles formed after the experimental intoxication, and the lesions continued to develop after the toxic feeding was stopped.

The harmful effect on dental calcification is, therefore, a delayed one, obviously due to an accumulated excess of fluorine, which has an indirect action through the medium of the endocrine glands upon the teeth and which leads to decalcification.

RUPTURE OF THE APEX OF THE CECUM FROM PARTURITION. Dr. Golvan. Rec. de Méd. Vét., cxiv (1938), pp. 788-789.

In 13 years of practice the author encountered four cases of rupture of the apex of the cecum, occurring during the act of parturition in cows. In three of the cases the accident was manifested by bloating, moaning and apparent prolapse of the rectum. In the other case the fetus was blocked in the pelvis. All of them were posterior presentations of abnormally large fetuses, with the feet still within the pelvic path. In each instance, forced delivery with ropes was required. Although the extraction was not particularly difficult, the floating end of the cecum, which had been forced into the rectum, burst with a crashing sound and spilled its contents over the operator.

How does this accident occur? It is sufficient to recall that the free end of the cecum can float backward into the pelvic cavity and, there, become blocked by the legs, hocks and thighs of the posteriorly presented fetus. As the fetus fills the pelvic path more and more, the end of the trapped cecum becomes a veritable balloon which is pushed backward, farther and farther, as the delivery proceeds. At a given moment, when the fetus completely fills the pelvic path, the content of the incarcerated cecum will burst through the ceco-rectal wall, or the cecum itself may herniate and present itself through the anus. The accident may occur without presenting these external manifestations; it may be detected only at the autopsy.

THE FINANCING OF VETERINARY SANITARY INSPECTION. (Title translated.) G. Thieulin. Rec. de Méd. Vét., cxiv (1938), pp. 769-787.

The author quotes figures, set down in various laws, which show how taxes are assessed, at so much per head or so much per kilo, to defray the expense of veterinary sanitary inspection. The intention of sanitary inspection of live stock and of animal products is to supervise the prophy-

laxis of contagious diseases of animals and of diseases, contagious or not, that are transmissible to man or other animals through the intermediary of their food supply.

In this rôle, the veterinarian is a hygienist of the first rank, and the means of financing his inspections is a matter of vital importance, local in practice but national in effect. It comprises the supervision of native and imported animals and products of animal origin, abattoirs, fairs, markets, transportation, refrigeration and all situations affecting the health of animals and the wholesomeness of food derived from them.

While the money collected by communities, municipalities and the state runs into large sums, the general plan is not satisfactory. It lacks the central control required for effective and uniform results. In short, veterinary sanitary inspection concerns the entire population and, therefore, should be financed from national revenues, and not by assessing the owner of the property inspected.

DURATION OF TETANIC TOXOID IMMUNITY. Ramon and Lemetayer. Rev. de Méd. Vét., xc (Jan., 1938), p. 45.

The vaccination of army horses against tetanus, begun in 1928, now includes 50,000 head. The technic consisted of two injections of 10 cc. each of tetanus toxoid to which tapioca had been added. Two-thirds of these animals received a third injection of 10 cc. after a considerable length of time (about a year) as an additional reinforcement. A check for the amount of specific antitoxin contained in the blood of vaccinated animals (selected here and there) after several years gave an average antitoxic titre of from 1/10 to 1 unit. It is known that the presence of 1/1,000 unit is sufficient to protect a horse against tetanus infection. The statistics of the army show that the morbidity and mortality from tetanus was absolutely nil among the horses that received the third dose.

SARCOPTIC MANGE: CONTAGIOUSITY FOR DOGS AND MAN. L. Pigoury and E. Nemeh. Rev. de Méd. Vét., xc (Jan., 1938), p. 38.

Man is receptive to the canine variety of *Sarcoptes scabiei*. It seems that the chances of contaminations vary with the virulence of the parasite. The period of incubation is about the same as in the dog, that is to say, 15 days, which is somewhat longer than in human mange. In man the clinical symptoms approach those caused by *Sarcoptes minor* of the dog and cat. The affection has a different physiognomy from that of the human-mange mite and is somewhat less tenacious, but nevertheless requires serious treatment.

DAS BANDAGIEREN DER PFERDE. (Bandaging of horses.) Langer. Zeit. f. Veterinärk., li (1939), p. 1.

When bandaging horses, the following general rules are to be observed: The individual tours of the bandage must be laid smoothly and without any folds. After the bandage has been applied, it should be possible to insert a finger between the upper rolls of the bandage and the leg. It is better to have the tours put on firmly, although tied loosely, than to have them placed loosely and attempt to tie the bandage firmly. New flannel bandages are to be soaked in cold water first, and dried before use. Tie-strings are to be flattened out well; damaged strings are to be replaced. The bandages are never to be tied either at the upper or lower end of the tours, since they will slip and might injure the skin by chafing.

The last turn of the bandage must be made about two or three widths distant from the end of the bandage. While the strings are being tied, they must not be made to lie upon the flexor tendon of the second phalanx or upon the canon bone. Bandages which are to be applied wet must be soaked previous to application. When the horse returns from work, the bandages are to be removed at once; the skin is to be examined for abrasions. Bandages are

not to be left on over night. Wet packs should never be applied longer than three days. After the bandages are taken off, they are to be cleaned and repaired.

FERRETS IN DISTEMPER-SERUM PRODUCTION. P. Goret. (Essai de Traitment de la Maladie de Carré par un Serum Specifique.) Rev. de Méd. Vét., xc (Jan., 1938), p. 47.

Goret, confronted with the difficulty of obtaining dogs perfectly sound and sensitive to the virus of Carré, uses ferrets as the producers of the virus, and adult dogs refractory to the disease for producing the serum. The dogs receive 10 cc. of a 20 per cent emulsion of the spleen of ferrets killed in the last stage of the disease. The dogs are "bled white" seven days later and the serum of the various dogs used is mixed together. Titering by the complement-fixation test shows that the blood serum is rich in antibodies.

Out of 27 affected dogs treated with this serum, 19 were cured and six died. (Two were not accounted for.) This serum, employed before the appearance of pulmonary complications, appears to be the best treatment at one's disposal. The doses recommended are 10 cc. subcutaneously and 10 cc. intramuscularly. An additional dose may be given subsequently. In complicated cases the serum treatment remains a precious adjuvant to the symptomatic treatment.

THERAPIE DER LUMBAGO. (Treatment of Myoglobinemia.) Tillmanns. Zeit. f. Veterinärk., li (1939), p. 49.

Previously advocated treatments for paralytic myoglobinemia of horses failed to bring about satisfactory recovery, especially in severe and moderately severe cases. According to our present day etiological knowledge, calcium therapy seems to be the most successful treatment. Even very severe cases of myoglobinemia will recover under calcium treatment, provided the treatment has not been delayed too long. The doses recommended in the literature have to be exceeded considerably for best results, but no complications will arise from this. Should a single intravenous injection of calcium prove to be insufficient, then the injection must be repeated the following day with the same or a smaller dose, until recovery is accomplished. The parenteral injection may be combined with oral administration of calcium.

CONTRIBUTION TO NATURAL MATERNAL NOURISHMENT IN SHEEP. (Title translated.) Prof. P. Pons. Rev. de Méd. Vét., xc (1938), pp. 593-618.

The author undertook to calculate with as much percision as possible the quantity of milk lambs suckle daily and its daily effect on growth. All other nourishment was withheld and the experimental lambs, kept apart from their mothers except for suckling, were weighed immediately before and after each suckling on a set of scales prepared for the purpose. The results are registered on seven tables and twelve graphs meticulously arranged to show:

1. The relations of gains to the quantity of milk ingested.

2. The proportion of nutritive principles laid down in the tissues in their relation to the quantity absorbed.

3. The loss represented in fecal and urinary elimination.

4. The potential energy in the new tissues and the relation of that energy to the potential energy of the alimentation.

5. Energy expended for upkeep and growth during the experiment.

A critical study of the descriptive text, the tables and the graphs reveals important information for those interested in that detail of ovine husbandry and superimposes new knowledge on the lactiferous nutrition of the newborn. The author establishes a correct relation between the quantity of milk consumed and the gains made by lambs during the first six weeks of their life, where no other nourishment is given.

BOOK NOTICES

AVIAN TUBERCULOSIS INFECTIONS. Wm. H. Feldman, D.V.M., M.S., Associate Professor of Comparative Pathology, Mayo Foundation for Experimental Education and Research, Graduate School, University of Minnesota. 482 pages, illustrated. The Williams and Wilkins Company, Baltimore, 1938. Price, $7.00.

The 482 pages of solid reading matter on but one type of the *Mycobacterium tuberculosis* might seem to contain, on first glance, a superfluity of material on a limited field of study. Yet the author was able to extend this book to 13 chapters, which shows the ground he was prompted to cover. This alone illustrates the ramifications of the tiny being we know as the "avian type" of the genus Mycobacterium.

In the initial chapter, entitled "General Considerations" the magnitude of the poultry industry in the United States at present is mentioned as one reason for setting down the known facts about this microörganism. The number of chickens in the United States is given as 454,000,000 and their value as $200,000,000; in all a $2,000,000,000 asset of the American farmer which grew out of a few chickens imported by Captain John Smith early in the 17th century.

The disease was first recognized as tuberculosis in 1868—14 years before Koch isolated the tubercle bacillus. This 32-page chapter, which includes a bibliography of 99 references, is a fascinating chronological story of events from Villeman in 1868 to Van Es and Schalk in 1914 and Van Es and Martin in 1930. In this sketch the author reproduces the well known map showing the geographic distribution of avian tuberculosis in the United States which strangely contrasts the incidence of the disease in various regions, the rather abrupt boundaries of which no one seems to stop and explain.

The remaining chapters comprise the following considerations of the subject:

II. Biological Characteristics.

III. Isolation and Culture.

IV. Diagnosis in Chickens.

V. Laboratory Procedure for Demonstrating Tuberculosis in Mammals and Fowls.

VI. Pathologic Anatomy in Chickens.

VII. Experimental Tuberculosis in Chickens.

VIII. Pathogenicity for Fowls Other Than Chickens.

IX. Pathogenicity for Mammals Other Than Man.

X. Pathogenicity for Cattle and Swine.

XI. Pathogenicity for Human Beings.

XII. Tuberculin and Tuberculin Test.

XIII. Dissemination and Control.

The illustrations reproducing fine photographic and photomicrographic work, the references at the end of each chapter, and the ten and a half pages of indexed subjects interspersed through the context of these chapters, make up a precious contribution to medical literature. When in the future the subject of avian tuberculosis is broached one will be thankful to have this book within reach.

VETERINARY HELMINTHOLOGY AND ENTOMOLOGY.. H. O. Monnig, B.A., D.Phil., B.V.Sc., Professor of Parasitology, Faculty of Veterinary Science, University of Pretoria. 2nd edition. 409 pages, illustrated. William Wood & Company, Baltimore. 1938.

The first edition of this work appeared in 1934, at which time it was received with considerable enthusiasm since, with the exception of Neuman's excellent work of 1892

and volume V of Law's *Veterinary Medicine*, no book covering the whole scope of helminthology had yet been published. In this country this literature was limited to texts in the form of articles published in current journals, college notes, and commercial house organs and bulletins. For that reason Monnig's work was a welcome arrival. It brought the known facts about helminths and arthropods between the covers of a well bound tome which, when checked against errors and omissions, was not found to be wanting in any serious respect.

The volume is divided into four sections, three on helminths and one section of 124 pages on arthropods. It is, therefore, two-thirds helminthology and one-third entomology, well illustrated, well written and conveniently arranged. The author dwells in detail on life cycles, methods of control, pathogenicity and medical treatment in regard to both worms and insects. Because these considerations are brought up to date, the book becomes valuable to practitioners as well as to students and teachers of parasitology.

The author closes this excellent work with a unique feature, a "Host Parasite List," ten pages long, in which is named and indexed each worm and insect commonly affecting the various species of hosts described in the text. Cattle lead with 118 helminths and arthropods; horses have 56, swine 50, man 58, etc., through the list of hosts generally included in a work of this sort. The list also enables the reader to determine at a glance the different hosts affected by the given parasites.

A GUIDE TO VETERINARY PARASITOLOGY AND ENTOMOLOGY. T. Southwell, D.Sc., PhD., A.R.C.Sc., F.Z.S., F.R.S.E., School of Tropical Medicine, University of Liverpool, and A. Kirshner, M.D., Ch.B., D.T.M., F.Z.S., formerly, School of Tropical Medicine, University of Liverpool. 2nd edition. 178 pages with 123 illustrations, 12 diagrams illustrating life histories. H. K. Lewis & Co., Ltd., London, 1938. Price, 8 shillings, net.

This small work contains lectures in condensed form which have been given to the veterinary students at the School of Tropical Medicine in the University of Liverpool. Lengthy descriptions are avoided and important points on diagnosis are clearly stated. It contains the orthodox classification of helminthic and arthropodic parasites commonly injurious to live stock and pet animals, gives the known facts about life cycles and habits, methods of diagnosis, and pathogenicity, but leaves the reader to plan his own therapeutics, notwithstanding that the mention of the specific medicinal agents now generally approved would add value to the work, for, after all, the only purpose of pathogenic parasitology is to plan means of exterminating parasites. But this omission is compensated in part by the excellent diagrams on life histories and the technics of microscopic fecal examinations.

COTTONSEED MEAL: ORIGIN, HISTORY, RESEARCH. Robert C. Curtis, North Carolina College of Agriculture and Experiment Station, coöperating with the United States Department of Agriculture. 502 pages, with 160 illustrations. The Robert Curtis Publishing Company, Raleigh, N. C., 1938.

The book is designed to promote the feeding of cottonseed meal, onetime waste product of cotton culture, but now a valuable component of the ration of farm animals and soil fertilizers. The value of cottonseed meal as a feed constituent lies in its high percentage of adequate protein, little of which was known before 1894, when, owing to a drought, large quantities were shipped to Iowa to replace corn in the feeding of beef cattle. The production of cottonseed is shown in a table covering the period from 1875 to 1927. For 1875 the amount is given as 1,687,000 tons valued at $2,530,000; for 1927 the amount produced

was 7,989,000 tons valued at $240,284,000. Cottonseed furnishes a valuable commodity of export and is thus an important factor in maintaining agricultural prosperity in the South.

The book treats in detail the use of cottonseed meal in the proper ratio with other feeds, such as corn-plant silage and other roughage and grain concentrates, and their effects on gains, milk production and reproduction.

The chapter on gossypol, the toxic ingredient of cottonseed and cottonseed meal, is of great interest to the veterinarian, who should be aware of the extent of the danger and where it lies. The author declares that gossypol in the meal, according to present knowledge, is not physiologically active. The amount contained in the meal is too small to be injurious to live stock.

The book contains reports of many well controlled feeding experiments conducted by capable authorities who thus make known the whole truth about this now important feed for farm animals. The tables are enlightening, the illustrations graphic, and the references so numerous as to satisfy anyone seeking further details of the subject.

PRINCIPLES OF VETERINARY SCIENCE. B. F. Hadley, Professor of Veterinary Science, University of Wisconsin, and Veterinarian of Wisconsin Agricultural Experiment Station. 3rd edition. 595 pages, illustrated. W. B. Saunders & Company, Philadelphia, 1939.

Since veterinary science is an established branch in the curriculum of the agricultural colleges, it naturally follows that a textbook limited to the part and depth of the study required to round out an agricultural course is a necessary piece of literature. Because veterinary science in the agricultural colleges and the veterinary profession were born at the same time in this country and grew up side by side, there was a time when it was difficult to remain faithful to one without offending the other.

While there were but few college-trained veterinarians to service our vast live stock industry, the teaching of medicine in the agricultural college was a rational step, a necessity, intended to qualify farmers in the treatment of sick animals. But with the development of an adequate veterinary educational system, it is remarkable how well the two came to a mutual understanding of their respective fields. Moreover, a glance through the annals shows that the teachers of veterinary science to the "ag students" have been active participants in national and state societies and pioneers in the development of a veterinary profession, not to mention their valuable contributions to periodical literature.

Hadley's *Principles of Veterinary Science* is a book that has followed the trend. It teaches what everyone entrusted with the care of farm animals should know, fundamentally, to become a capable husbandman, qualified in prevention and first aid and knowing when and where to seek expert advice and service. In fact, one of the pleasant contemplations of this hour is the fine understanding which exists between the chair of veterinary science in the agricultural college and the regular veterinary college curriculum which such books have helped to build.

Part I treats of anatomy and physiology, in 257 pages, and part II of animal disease in such a fashion as to impart a broad knowledge of veterinary practice without danger of curtailing the prerogatives of the legitimate practitioner. In this respect, Hadley's work is refreshing. In fact, it contains many a paragraph worthy of considerable praise for frank expression and perhaps, also, for artfully dodging the rough spots of unknown etiology. In reading the work from cover to cover, looking for a place for a reviewer to exercise his right to criticize, one wonders how an author can collect that amount of up-to-date knowledge on so many diversified subjects without "stubbing his toes."

THE NEWS

OF GENERAL INTEREST

New York World's Fair Exhibit

The portrayal of modern developments in veterinary science combined with oustanding achievements of the past will greet the 60 million visitors who will attend the New York World's Fair, to open, as scheduled, on April 30. Occupying a prominent space in the Medical and Public Health Building, centrally located on Constitution Mall under the shadows of the Trylon and Perisphere, the exhibit of the American Veterinary Medical Association, now approaching completion, utilizes motion, light, and color in telling a graphic story of service to man and animals. The conquest over disease, with resulting benefits to the country's live stock resources and public welfare, receives principal emphasis. This line of approach, those in charge of the exhibit believe, is more impressive to the majority of visitors at a large fair than technical material would be.

On entering the exhibit space, in the form of a bay about 22 feet wide and 30 feet deep, the observer sees, on his left, a rotating cylinder showing flying insects. The names of diseases carried by insects also appear on the cylinder. This unit illustrates also the classical veterinary discovery of the manner in which tick fever is spread, which was the pioneer work that proved insects carry disease. Malaria, yellow fever, typhus fever and bubonic plague are named as examples of other maladies transmitted in similar manner. Three-dimensional models show the greater quantities of milk and meat supplied by tick-free cattle than by tick-infested ones. There is also a working model of a dipping vat in operation.

The visitor next observes a display illustrating how only healthy cows can produce safe and wholesome milk supplies and why, in this public health activity, veterinary supervision is essential. This unit features the effective measures used in curbing bovine tuberculosis, Bang's disease, and mastitis. It illustrates also types of veterinary inspection, various tests, and the disposal of unsound animals. A panel showing the reverse situation, namely, the transmission of disease from man to cow, is also included. A device involving the flashing of green and red lights portrays the great reduction in bovine tuberculosis in the United States during the period 1924-1938.

In a conspicuous place along the side of the exhibit, the visitor next observes the effects of equine sleeping sickness, or encephalomyelitis. The display presents the increasing trend of this malady in the United States during the last four years and the discovery, during 1938, of its transmissibility to man and of the efficacy of the newly developed embryonic chick vaccine in combating these equine losses.

Adjoining the above unit is a semicircular alcove containing a photodiorama of a modern veterinary hospital for small animals. The observer sees, in cross section, the rooms and equipment used for reception, examination, x-ray, electro-therapy, surgery, laboratory diagnosis, and other purposes. In addition, a moving device shows different breeds of dogs on leash entering the hospital sick and leaving, on the other side, cured.

The visitor next witnesses the conquest of veterinary science over hog cholera. This achievement is shown in terms of both animals saved and money losses prevented, together with information on the scope of the anti-hog-cholera serum industry that has developed as a sequel to the discovery of anti-hog-cholera serum. This is the first virus disease in this country for which a serum has been made generally available by the serum producers.

The final feature of the exhibit is a large display illustrating the scope of the meat industry, the thoroughness and economy of federal meat inspection, and the important rôle of meat in the diet. Here, the visitor learns that approximately 70,000,000 food animals receive federal inspection annually, also that the vast majority of these animals are healthy—condemnations being a small fraction of 1 per cent.

In connection with illuminated charts showing the food value of meat, visitors learn how to select a normal diet, a reducing diet, or a gaining diet, as desired. These are conveniently shown as moulages in natural colors on three moving tables.

The entire exhibit on veterinary medicine and public health extends along approximately 60 lineal feet of wall space and includes 590 square feet of floor area. The exhibit is sponsored by the American Veterinary Medical Association with the generous aid of the National Live Stock and Meat Board, Borden Company, Lederle Laboratories, Hog-Cholera Serum Producers, American Animal Hospital Association, and Friends of Pet Animals. In addition, various state, regional and similar veterinary associations and a few individuals have generously supported the exhibit financially and otherwise. Nevertheless, in order to complete the exhibit on the scale above indicated, more funds are necessary. Since the Fair is to run for 180 consecutive days, the lighting, motor-driving, cleaning, guarding, demonstrating, repairing, and insuring this display will be constant items of expense.

The Committee responsible for planning the exhibit, in consulation with World's Fair officials and technical experts, includes R. S. Childs, C. E. DeCamp, R. F. Eagle, Adolph Eichhorn, W. A. Hagan, R. S. MacKellar, Jr., D. H. Udall, Cassius Way, C. P. Zepp, and Chairman John R. Mohler. Besides planning the exhibits, the committee has made arrangements for including motion-picture films on veterinary and live stock subjects in connection with motion-picture programs to be shown at the Fair.

J. R. M.

Exhibit Fund

John R. Mohler, chairman of the special committee on the veterinary educational exhibit at the coming New York World's Fair, reports the following contributions to the fund being raised to finance this project:

Dallas-Fort Worth Veterinary Medical Association	$ 15.00
Illinois Veterinary Medical Association	200.00
Indiana Veterinary Medical Association	50.00
Intermountain Livestock Sanitary Association	50.00

These contributions, added to the $2,108.20 already subscribed, bring the total to $2,423.20.

A. V. M. A. Publicity Plans

Plans for the 1939 publicity of the American Veterinary Medical Association have been drawn up and submitted to the Executive Board of the A. V. M. A.

As outlined, the 1939 campaign will have three general objectives, which are: (1) to increase the business of present members; (2) to stimulate advance interest in the Memphis convention in order to assure a large attendance and to obtain wide publicity for this event, and (3) to spotlight wide public attention upon the veterinary exhibit at the New York World's Fair.

In an effort to aid in increasing the membership of the Association and, at the same time to increase the business of present members, the publicity will be aimed toward establishing certain definite facts in the public's mind, namely: (1) that members of the A.V.M.A. are the most outstanding members of the profession; (2) that members of the A.V.M.A. are the leaders in the development of scientific advances in veterinary medicine and (3) that members of the A.V.M.A. are skilled practitioners in the care of animal diseases.

New stories, feature stories and radio talks are being planned to carry these facts to the public.

It will be stressed repeatedly that the public is certain to receive the highest ethical veterinary service when it patronizes members of the A.V.M.A. Stories also will be released dealing with the scientific advances made by members of the Association in the care and treatment of animals. Moreover, news releases will depict the activities of A.V.M.A. members and, of course, the man's membership in the Association will be stressed in each story. This publicity should help the individual locally and the Association nationally.

The Memphis meeting will be given vigorous publicity promotion during the months preceding it in order to stimulate maximum attendance. News stories and radio talks are already being planned to focus interest on the convention proceedings. It is expected that these stories and talks will attract the attention of editors and that the publicity total will equal, if not exceed, the record publicity total obtained for the Diamond Jubilee Convention in New York City.

Efforts will be made to release news stories about every man attending, and every talk at the meeting will receive fullest publicity attention.

The veterinary exhibit at the New York World's Fair is expected to be of great value in acquainting the public with the valuable public health services of the veterinary profession. News items will be released prior to the opening of the Fair which will inform future visitors fully about the exhibit and, when the Fair opens, there will be special stories from time to time to stimulate interest in the exhibit.

World's Poultry Congress

The Seventh World's Poultry Congress and Exposition will be held at Cleveland, Ohio, July 28 to August 7, as previously announced. As the date of the Congress approaches, movements to stimulate interest in this outstanding event among veterinarians have been started by a number of poultry pathologists, practitioners and others who aim to bring the poultry industry and the veterinary service into closer working relations. That the one has neglected to court a closer attention to the other in the past is not assailable and, as a sequence, both have been large-scale losers. The poultry industry goes on suffering tremendous losses from disease and nutritional disorder and the veterinary service as a whole continues to be a somewhat uninterested bystander, instead of an ambitious confederate.

Among the leaders in these movements is the Ohio State Veterinary Medical Association, which has appointed a special committee charged with the task of bringing the interesting program of the Congress to the attention of the veterinary profession. O. V. Brumley, Dean of the College of Veterinary Medicine, Ohio State University, past president of the A.V.M.A., in sponsoring the publicity work, invites attention to the opportunity the Congress affords in this connection. He hopes for a large veterinary representation.

The preliminary exposition in Washington, July 25-27, the Field Day of the Congress at the Ohio Agricultural Experiment Station, at Wooster, Ohio, and the special features of the Congress in Cleveland will be described in detail in the May and June issues of the JOURNAL.

Ohio State Veterinary Conference

The Twelfth Annual Conference of the College of Veterinary Medicine, The Ohio State University, will be held June 14-16, 1939. This year, as formerly, the program is quite an extensive one and includes some of the foremost veterinarians and educators in the country. The entire three-day period will be devoted to the discussion of topics of vital importance to every veterinarian. A special effort has been made in arranging the details of the program so that its quality and variety will appeal to all.

The program this year has been formulated for the purpose of giving to the practitioner the very latest developments in veterinary medicine. It is generally conceded that such intensive short courses provide these educational opportunities.

In past years, the attendance has been very gratifying to those who have been interested in promulgating these courses. The interest manifested in the programs and the various discussions entered into by those in attendance also have been a source of much gratification. An invitation is hereby extended to all veterinarians to be present on this occasion.

Programs of the conference will be mailed in the near future. In looking over the program you will observe the names of many experts nationally known in their specific lines of work, assuring you of an excellent and instructive program during the entire period.

Remember the dates, June 14, 15 and 16, 1939.

COMING MEETINGS

Small Animal Hospital Association, Los Angeles, California. April 4, 1939. R. W. Gerry, Secretary, 8474 Melrose Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association Of. Hotel New Yorker, New York City. April 5, 1939. C. R. Schroeder, Secretary, New York Zoölogical Park, Bronx, N. Y.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. April 6, 1939. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. April 6, 1939. W. T. Hufnall, Secretary, 1612-14 E. Alabama Ave., Houston, Texas.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. April 11, 1939. W. A. Young, Secretary, 157 W. Grand Ave. Chicago, Ill.

Maine Veterinary Medical Association. Orono, Me. April 12, 1939. A. E. Coombs, Secretary, 1 Kennebec St., Skowhegan, Me.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. April 12, 1939. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. April 12, 1939. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Kansas City Veterinary Medical Association. Kansas City, Mo. April 17, 1939. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. April 17, 1939. Glenn A. Tucker, Secretary, Vista, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. April 19, 1939. Charles Eastman, Secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Southern Alabama-West Florida Veterinary Medical Club. Brewton, Ala. April 20, 1939. E. M. Nighbert, Secretary, Cantonment, Fla.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. April 26, 1939. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. April 26, 1939. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Connecticut Veterinary Medical Association. G. Leroy Cheney's Hospital, Woodbridge, Conn. May 3, 1939. George E. Corwin, Secretary, State Office Building, Hartford, Conn.

Ohio State University Annual Veterinary Conference. Ohio State University, Columbus, Ohio. June 14-16, 1939. O. V. Brumley, Dean, Ohio State University, Columbus, Ohio.

Missouri State Veterinary Medical Association. Saint Louis, Mo., June 15-16, 1939. C. L. Campbell, Secretary, 7224 Tulane St., Saint Louis, Mo.

American Association for the Advancement of Science. Milwaukee, Wis., June 19-24, 1939. F. R. Moulton, Secretary, Smithsonian Institution Building, Washington, D. C.

California State University Medical Association. Santa Barbara, Calif., June 26-28, 1939. Chas. J. Parshall, Secretary, 319 B Street, Petaluma, Calif.

World's Poultry Congress, Seventh Annual. Cleveland, Ohio. July 28-August 7, 1939. W. R. Hinshaw, Chairman, Section on Pathology and Disease Control, University Farm, Davis, Calif.

American Veterinary Medical Association. Memphis, Tenn., August 28-September 1, 1939. L. A. Merillat, Executive Secretary, 221 N. LaSalle St., Chicago, Ill.

State Board Examination

Nebraska Board of Veterinary Examiners. June 26-27, 1939. Further information may be obtained from Mrs. Clark Perkins, Director, Bureau of Examining Boards, State Capitol Bldg., Lincoln, Neb.

MEETING REPORTS

Cornell's Thirty-first Annual Conference for Veterinarians

The annual Cornell Conference for Veterinarians was held on January 12 and 13, 1939, at the New York State Veterinary College, Ithaca, New York. There was a registration of 330. Allowing for at least 50 who did not register, it is safe to say that at least 380 veterinarians attended. It is interesting to note that nearly all classes graduated from the Cornell School were represented. Of the registered veterinarians, 288 were graduates from the New York State Veterinary College at Cornell University.

A general session was held Thursday morning. Two sectional meetings were held Thursday afternoon. Five papers on small animal diseases were presented at one afternoon session and five papers on bovine diseases at the other. Thursday evening was occupied by alumni meetings and inspection of the Veranus A. Moore Laboratory. Clinics and demonstrations were held Friday morning. Ten different demonstrations and clinics were conducted throughout the morning session. The general session Friday afternoon and the banquet Friday evening completed the program.

The men outside of the New York State Veterinary College faculty, who assisted with the program were: Dr. Carl F. Schlotthauer, The Mayo Foundation for Education and Research, Rochester, Minnesota, Dr. F. D. Holford, Chief, Veterinary Department, Borden Farm Products Co., New York City, Dr. J. A. Henderson, New Jersey Holstein Breeders Coöperative Association, Dr. B. M. Lyon, Lederle Laboratories, Inc., Pearl River, New York, Dr. Leo P. Larkin, Roentgenologist, Ithaca, New York, Dr. S. A. Johnson, Kinderhook, New York, Lieut. Col. C. W. Greenlee, Veterinary Corps, Governor's Island, New York, H. Wallace Peters, Provost of Cornell University, Walter King Stone, Professor of Architecture, Cornell University, and W. I. Myers, Professor of Farm Finance and Head of the Department of Agricultural Economics, Cornell University, formerly Governor of the Farm Credit Administration, Washington, D. C.

E. L. Brunett, *Reporter.*

Houston Veterinary Association

On Thursday, March 2, 1939, at the regular monthly meeting of the Houston Veterinary Association, held at Houston, Texas, the following members were elected to office: Madero N. Bader, president; Don B. Strickler, vice-president; W. T. Hufnall, secretary, and J. Gilbert Horning, corresponding secretary.

An article on "The Common Dog Flea" was read by J. Gilbert Horning, and William James showed an interesting movie on "The Proper Technic for De-Skunking a Skunk," or, in other words, removing the musk sacks of a pole-cat. The usual questionnaire was issued. Don B. Strickler was the first-prize winner. This monthly questionnaire is looked forward to by the men and is interesting competition as well as quite educational As the one who has to get up the questions gets plenty of time for study and all questions have to be confined to a strictly professional nature, it takes an interesting evening of one's time to compile them. The winner of each quiz has to get up the questions for the following meeting.

J. Gilbert Horning,
Corresponding Secretary.

Connecticut Veterinary Medical Association

On February 1, 1939, the Connecticut Veterinary Medical Association held its annual meeting at the Hotel Bond, Hartford.

S. W. Stiles, of Falmouth Foreside, Maine, spoke on "Short Cuts in Hospital Practice" and Richard N. Shaw of Northboro, Mass., talked on "Artificial Insemination and Some Major Breeding Troubles."

Officers for the ensuing year were elected as follows: President, F. F. Bushnell, of Manchester; first vice-president, F. I. Maxon of Hartford; second vice-president, A. T. Gilyard, of Waterbury, and secretary-treasurer, Geo. E. Corwin (reëlected). J. M. Curry, of Hartford, is the only new member of the Board of Censors, G. Leroy Cheney, of Woodbridge; G. H. Ludins, of Hartford; N. W. Pieper, of Middletown, and J. J. Kavanek, of Hartford, having been re-elected.

Geo. E. Corwin, *Secretary.*

Illinois State Veterinary Medical Association

The fifty-seventh Annual Meeting of the Illinois State Veterinary Medical Association was held at the Leland Hotel, Springfield, February 16-17, 1939, with approximately 300 veterinarians and their wives in attendance. Following the President's address, the following literary program was presented:

"Community Sales"—H. C. Rinehart, Springfield.

"Some of the Responsibilities of the Veterinary Profession in Animal Disease Control"—J. R. Brown, Ottawa.

"Calcium Therapy"—L. N. Morin, Clinton.

"Veterinary Medical Organizations"—H. D. Bergman, Ames, Iowa.

"Cattle Practice"—C. R. Donham, Ohio State University.

"Recently Acquired Knowledge Concerning Equine Encephalomyelitis and Its Relationship to the Problem of Control"—W. S. Gochenour, Zionsville, Ind.

"Encephalomyelitis of Horses" — A. H. Schmidt, Triumph, Minn.

"A Condition in Cattle Contributed to Plant Poisoning"—L. A. Dykstra, Lena.

"Cineographic Views of Cases at the Summit Dog and Cat Hospital"—Joseph B. Engle, Summit, N. J.

"Canine Practice by the General Practitioner"—J. V. Lacroix, Evanston.

"Diseases of Newborn, Suckling and Weanling Pigs"—H. C. H. Kernkamp, St. Paul, Minn.

"Source of Infection on Cholera-Infected Farms in Illinois"—J. H. Murphy, U. S. Bureau of Animal Industry, Paris.

"Enteritis of Swine"—L. A. Gray, Bushnell.

The dinner session was given over entirely to a banquet, entertainment and dancing. The highlight of the meeting was the excellent address by Dr. Bergman in which he emphasized the necessity of every veterinarian supporting his state and national associations.

The following officers were elected: W. B. Holmes, president; J. W. Lucas, of Abingdon, vice-president; R. M. Carter, of Alexis, member of the Executive Board.

C. C. HASTINGS, *Secretary.*

Society of Phi Zeta

At a meeting of Alpha Chapter of Phi Zeta, an honorary society in veterinary medicine, on March 3, 1939, the following were elected to active membership:

Class of 1939:

Daniel Paul Sasmore
James Joseph McCarthy
Raymond Fagan
Alexander Donald Rankin
Lyndon Wainwright Potter
Gerald Almon Faatz

Class of 1940:

James Andrew Baker
Ralph Eugene Loomis
Rebecca Gifford
Ralph Louis Gangarosa

At the same meeting Cassius Way, of New York City, and James S. Carnrite, of Fort Plain, N. Y., were elected to honorary membership.

W. S. STONE, *Secretary Pro Tem.*

Manitoba Veterinary Association

The forty-ninth annual meeting of the Veterinary Association of Manitoba was held in the Marlborough Hotel, Winnipeg, on February 16, 1939. The meeting was attended by more than 50 veterinarians, many of them practitioners from country points.

The president, F. M. Coombs, of Hamiota, Manitoba, in his opening address, discussed the events of interest to the profession during the past year.

The morning session was taken up with a discussion of Association affairs. W. Hilton, Secretary-Treasurer and Registrar, in his report welcomed four new members to the Association during the past year.

Officers for 1939 were elected as follows: President, J. A. Allen, of Winnipeg; vice-president, D. J. Lawson, Shoal Lake; secretary-treasurer and registrar, W. Hilton, Winnipeg. In addition, the following members were elected to the Council: F. M. Coombs, Hamiota; E. L. Houck, Hartney; R. H. Lay, Winnipeg, and A. Savage, Winnipeg.

The newly-elected president, J. A. Allen, occupied the chair during the afternoon session which was opened by an address by H. H. Ross, of Brandon, on the subject, "More Jurisprudence in Veterinary Practice."

J. M. Brown, Professor of Animal Husbandry at the University of Manitoba, discussed the work being done at the University in the matter of "Artificial Insemination."

"The Present Status of Equine Encephalomyelitis and Its Control," was the topic of a paper presented by B. M. Lyon, assistant director of Lederle Laboratories, Pearl River, N. Y. The general discussion which followed brought out some interesting phases in the preventive treatment of this malady. Dr. Lyon used a number of slides to illustrate his address.

Alfred Savage, Pathologist, University of Manitoba, spoke on the topic, "Figures, Fiction and Fancy," bringing the afternoon session to a close.

R. H. LAY,
Resident Secretary for Manitoba.

Kansas Veterinary Medical Association

The Kansas Veterinary Medical Association held its Thirty-fifth Annual Convention in Manhattan, February 15-16, 1939, with headquarters at the Hotel Wareham and program, both literary and clinic, at the Division of Veterinary Medicine, Kansas State College.

General attendance broke all records. This was made possible not only by the good weather but also from the type of program which was supplied. The program committee "sounded out" the membership by means of a postal card ballot as to the type of program which they wanted. It was purely a practitioners' program. All discussions were of a practical interest.

Speakers were Leonard Rosner, of St. Louis, who discussed "The Veterinarians as Milk and Dairy Inspectors." R. M. Hofferd, of Cedar Rapids, and Frank Breed, of Lincoln, Neb., discussed swine diseases in a very practical manner. L. M. Roderick, newly appointed head of the Pathology Department, Kansas State College, discussed "Sheep Practice." Professor V. D. Foltz presented a discussion on "Mastitis" and demonstrated the Hotis test for mastitis. A. D. Weber gave a very practical discussion on "Balancing Animal Rations." The equine encephalomyelitis discussion was led by M. P. Schlaegel, of Beloit, who presented case reports of all the patients under his care during the recent outbreak.

At the clinic session R. L. Anderes and Irvin Twiehaus, of Kansas City, Mo., demonstrated "Artificial Insemination." E. W. Theiss, of Independence, Mo., demonstrated his method of "Handling Fractures in Small Animals." Methods of anesthesia in small animals were demonstrated by R. L. Anderes. "Trichomoniasis and Histomoniasis Lesions in Turkeys and the Parasite," was demonstrated by M. J. Twiehaus.

The following officers were elected: President, J. A. Bogue, of Wichita; vice-president, Roy L. McConnell, of Coffeyville; to Board of Directors, Sam Hayes, of Hutchinson, and secretary-treasurer, Chas. W. Bower, reëlected. Topeka was chosen for the 1940 meeting place.

C. W. BOWER, *Secretary.*

U. S. GOVERNMENT

Regular Army Service

Lt. Colonel George L. Caldwell is relieved from assignment and duty in the office of the Surgeon General, Washington, D. C., effective on or about August 19, 1939, and assigned to Fort Riley, Kan., for duty.

Lt. Colonel Ralph B. Stewart is relieved from assignment and duty at Fort Riley, Kan., effective on or about June 1, 1939, and is assigned to duty as instructor and director, the Army Veterinary School, Army Medical Center, Washington, D. C.

The resignation of First Lieutenant Fred Lewis Herring is accepted by the President, effective February 10, 1939.

Lt. Colonel Claude F. Cox is relieved from duty at Fort Des Moines, Iowa, effective in time for him to proceed to New York, N. Y., and sail on transport scheduled to leave that port on or about June 1, 1939, for the Hawaiian Department, for duty.

Lt. Colonel Harry L. Watson is assigned to Fort Huachuca, Ariz., effective upon completion of his tour of foreign service in the Hawaiian Department.

Captain Russell McNellis is relieved from his present assignment and duty of the Army Medical Center, Washington, D. C., effective in time to proceed to New York, N. Y., and sail on transport scheduled to leave that port on or about June 1, 1939, for the Philippine Department and, upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

The War Department has announced a competitive examination to be held from July 24 to July 29, 1939, both dates inclusive, for the purpose of qualifying Doctors of Veterinary Medicine for appointment as First Lieutenant in the Veterinary Corps of the United States Army to fill contemplated vacancies.

Graduates of recognized veterinary colleges are eligible for the examination provided they are not less than 22 9/12 years of age at the time of examination and will not be over 32 years of age at the time it is possible to tender them a commission.

Applicants will be authorized to appear before examining boards convened at Army stations in representative sections of the United States to conduct the examination.

Complete information and application blank will be furnished any interested veterinarian upon request to The Adjutant General, War Department, Washington, D. C.

Applications for this examination will not be considered if received after July 1, 1939.

Veterinary Corps Reserve

NEW ACCEPTANCES

(First Lieutenants)

Au, Robert John, 6245 23rd, N. E., Seattle, Wash.

Ward, William Deere, Route 1, Box 377A, Tulare, Calif.

PROMOTIONS

To Lt. Colonel: John A. O'Connell, Room 616, 31 Milk St., Boston, Mass.

To Major: Philip H. Fulstow, 99 W. Main St., Norwalk, Ohio.

To Captain: Alta Roy Bruner, Oakland City, Ind.

To 1st Lieut.: Earnest Paul Bernard, Sabina, Ohio.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

1st Lieut. Don C. Vest, Fort Moultrie, S. C.

1st Lieut. Geo. F. Scheetz, West Virginia District, Charleston, W. Va.

1st Lieut. Willard R. Merchant, Presidio of San Francisco, Calif.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

1st Lieut. Samuel F. Huber, Schenectady, N. Y. 1st Lieut. Howard F. Carroll, Presidio of San Francisco, Calif.

B. A. I. Transfers

Daniel P. Arron (Wash. '36), from West Fargo, N. Dak., to Spokane, Wash., on meat inspection.

Clyde W. Bean (Colo. '35), from Nashville, Tenn., to Beltsville, Md., on Bang's disease.

Albert D. Conley (Ont. '21), from Mason City, Iowa, to Eau Claire, Wis., on meat inspection.

Nathan N. Crawford (Iowa '09), from Omaha, Neb., to Baltimore, Md., on meat inspection.

Melvin E. Hodgson (K.S.C. '32), from Wichita, Kan., to Oklahoma City, Okla., on meat inspection.

Vantriff S. Jacobi (McK. '18), Madison, Wis., from meat inspection to tuberculosis eradication.

Vernon K. Jensen (Ont. '33), from Milwaukee, Wis., to South Saint Paul, Minn., on meat inspection.

Otto L. Montgomery (Colo. '38), from Salt Lake City, Utah, to Portland, Ore., on Bang's disease.

Oliver W. Orson (Tex. '32), from Spokane, Wash., to Fort Worth, Texas, on meat inspection.

Raymond E. Pinkert (Ont. '36), from South Saint Paul Minn., to Milwaukee, Wis., on meat inspection.

Melvin M. Rabstein (U.P. '37), from Baltimore, Md., to College Park, Md., on Bang's disease.

PERSONAL NOTES

Births

To Dr. and Mrs. O. Norling-Christensen, of Wilmette, Ill., a son, Olof, February 17, 1939.

To Dr. and Mrs. E. G. Hughes, of Sleepy Eye, Minn., a son, James Emory Hughes, February 20, 1939.

To Captain and Mrs. R. A. Boyce, Jr., of Fort Sam Houston, Texas, a daughter, Elizabeth Hope, February 25, 1939.

Marriages

Basil Barclay (Mich. '38), of Detroit, Mich., to Beatrice Siebert, of Lansing, Mich., on February 17, 1939.

Robert L. Anderes (K.S.C. '34), of Kansas City, Mo., to Catherine Busey, on March 18, 1939, at Kansas City.

Activities

C. H. Clark (Mich. '29), of Lansing, Mich., was reappointed State Veterinarian on February 20, 1939.

Arthur J. Powell (Wash. '11), of Lewiston, Idaho, has been appointed Director of the Idaho Bureau of Animal Industry, succeeding T. A. Elliot (Wash. '14).

J. A. Winkler (Cin. '18), of Fort Thomas, Ky., was toastmaster at the annual banquet of the Campbell County (Ky.) Chamber of Commerce, held at Covington on March 7, 1939.

E. C. W. Schubel (U.S.C.V.S. '11), of Blissfield, Mich., addressed the Lanaway County (Mich.) Council of Agriculture recently on "The Relationship of the Veterinarian and the Extension Program."

John H. Gillman (St. Jos. '17), of Memphis, Tenn., underwent an operation for appendicitis in January, but he has recovered completely and is back at work again, busy with plans for the A.V.M.A. meeting in August.

I. E. Newson (K.C.V.C. '09), of Fort Collins, Colo., member of the A.V.M.A. Executive Board for District 6, has gone to Arizona for his health. The climate there is expected to benefit his heart ailment. He plans to return to Fort Collins just as soon as his health permits.

Walter A. Lawrence (Colo. '36), of San Antonio, Texas, recently acquired an acreage at the edge of the city limits, which was the home and hospital of the late E. V. Dunbar. The hospital has been modernized and new kennels have been constructed. His residence joins the hospital.

C. Arthur Boutelle (McGill '95), city veterinarian of Newton, Mass., has the "softest" job in the entire country, it is said. He was recently appointed to the unpaid post of slaughtering inspector for his residential community, an office required by law. But the law specifically forbids slaughtering in Newton.

J. P. West (McK. '07), of Madison, Wis., and T. H. Ferguson (Ont. '96), of Lake Geneva, Wis., representing the Wisconsin Society of Veterinary Graduates, appeared before the Committee on Agriculture of the state assembly on March 1. They pointed out the dangers involved in a proposed bill to distribute free hog-cholera serum to Wisconsin farmers. Ferguson commented: "It's the most foolish bill I have ever encountered and, from the point of protection to our domestic animals, I can see where only serious harm will result."

Chas. H. Haasjes (Gr. '18), of Shelby, Mich., studies law as a hobby and, during the rest of his leisure hours, writes jokes and collects witty sayings which would be of interest to veterinarians. Recently, he sent this note to the A.V.M.A. office: "The latest development in veterinary medicine in Michigan is that it is cheaper to rent pasture for bob-tailed horses than those with long tails. The horse with a long tail can switch the flies off with his tail and be eating grass all the time, while the bob-tailed horse has to swing his head to both sides of his body, and of course, can not eat while doing this."

George B. Hartke

George B. Hartke, of Cincinnati, Ohio, died on January 29, 1939. He was born in Cincinnati and graduated from the Cincinnati Veterinary College in 1917. Dr. Hartke joined the A.V.M.A. in 1918.

Jonathon E. Gibson

After an illness of eight weeks, Jonathon E. Gibson died on February 10, 1939, at his home in Indianapolis, Ind. He was born at Jamestown, Ind., on February 11, 1862. After graduating from the Indiana Veterinary College in 1904, Dr. Gibson served as postmaster at Jamestown for five years. Then he entered the service of the U. S. Bureau of Animal Industry, from which he was retired in 1933. He joined the A.V.M.A. in 1917.

Mark L. Miner

Mark L. Miner of Greenfield, Mass., died on October 9, 1938. He was born in Richford, Vt., on May 3, 1864. After graduating from the Chicago Veterinary College in 1889, Dr. Miner entered practice at Amesbury, Mass. About 1893, he removed to Greenfield, where he practiced until his death. From the standpoint of years of service Dr. Miner was the senior veterinarian of the state, having first acted as a *per diem* agent in 1895 of the department now designated the Division of Live Stock Disease Control. Later he became District Agent for the Department, serving in this capacity until his retirement in 1934.

Dr. Miner was a member of the Massachusetts Veterinary Association.

CLASSIFIED ADVERTISEMENTS

Books and Magazines

PICTURE CHART of all Breeds, $1; three for $2. Judy's *Dog Encyclopedia,* all dog subjects, $5. *Dog World* monthly, $2 for one year; $3 for two years; $5 for five years. JUDY PUBLISHING COMPANY, 3323 Michigan Blvd., Chicago.

For Sale

PRACTICES furnished and sold—all states. Positions furnished—all kinds. Assistants—all kinds furnished. Established 33 years. F. V. Kniest, 1537 So. 29th St., Omaha, Neb.

Position Wanted

EXPERIENCED veterinarian will consider deal, position, lease, or partnership. Will buy a practice. California or western states only. Address: "S," care of F. V. Kniest, 1537 So. 29th St., Omaha, Neb.

Wanted

THE LARYNX of a horse with bilateral recurrent nerve paralysis and also one with unilateral paralysis. Communicate with Dr. Brien T. King, Medical and Dental Building, Seattle, Wash.

The fossil remains of a Pleocene dog—age 8 million years—has been unearthed in the Mojave Desert.

A singing mouse in the home of Mrs. Beatrice Fenman, Chicago, chirps like a cricket and weaves a nest from strings as neatly as a Navajo Indian.

A nanny goat brought into court charged with emitting an odor offensive to the neighbors was acquitted by a Florida court when a teacher of zoölogy testified that only billy goats are offensive to the public nose.

A Memphis (Tenn.) swallow set fire to a church when it accidentally scratched a match carried to the steeple where it was building its nest. The first was automatically extinguished by the melting of icicles hanging above the nest.

There is no such breed of dogs as a rat terrier, says Bob Becker in the *Chicago Tribune.* The smooth fox terrier and the Manchester terrier are commonly thought of as "ratters," this connoiseur of dogs tells the readers of his popular column.

By carrying "Vote for Chaddock" on his dog jacket, Ginger, a Boston terrier, is credited with having won the election of his master to the office of registrar of deeds at Benton Harbor, Michigan—*Bob Becker.*

Among the diseases of animals, rabies makes the headlines of the secular press more often than other animal ailments, because no disease of man is more dreaded nor more dreadful, than this unfortunate infection of man's best friend.

B. D. Pope conducts a fish hospital in London, where diseases of the finny tribe are treated with all the skill of ordinary veterinary work. In *Pearson's* for November, 1938, Lyn Evans describes diseases of fish and methods of treatment comparable to the text of the best technical literature.

The Anti-Cruelty Society of Chicago has made the headlines all over the country for having induced the police department of that city to install dog kennels in police stations where dogs may be humanely housed until disposed of. W. A. Young, secretary of the Chicago Veterinary Medical Association, veterinarian for the Society, sponsored the innovation.

Elsewhere in this issue is an article on the treatment of a horse suffering from a tibial fracture, which recovered under the ingenious treatment of Dr. J. F. Thomas who deservedly made the headline of the newspapers for his successful handling of a case that is generally declared incurable.

Tailwagger, Hollywood's new dog magazine, quotes Emery Lukes, veterinarian of Wilkes-Barre, Pa., as having mercifully destroyed a dog that was 26 years old. The dog had been the lifetime companion of its owner, a young lady of the same age.

On February 5, 1939, Wild Animal Trainer Clyde Beatty reported the birth of lion quintuplets. Since the birth of the lion quints was as unusual as that of the Dionne quintuplets, Beatty explained, he decided to name the cubs, all females, after the famed Dionne children—Cecile, Yvonne, Marie, Annette, and Emilie.

In Buckingham, Pa., strange sounds emanated recently from the organ in historic Trinity Episcopal Church. An organ expert was called in, diagnosed the trouble as "chipmunkey business," and then proceeded to remove a family of eight chipmunks that had built a nest in the pipes. Now, the organ is in tune again.

Surgeons (human) perform close to 1,000,000 operations annually in the United States. As to kind, tonsillectomy stands first and setting fractures second, according to a survey made by S. D. Collins, of the United States Public Health Service, of 8,758 families living in 130 localities in 18 states.

Jen-Sal

Encephalomyelitis Vaccine
(Chick)

Vaccination in April, May or June with Encephalomyelitis Vaccine (Chick) is recommended by field veterinarians and research workers for the control of Encephalomyelitis in horses.

Breeders of purebred horses, owners of valuable race horses and hunters and horse owners everywhere have been encouraged to vaccinate all horses and mules with Encephalomyelitis Vaccine early.

Write for post cards and popular article on vaccination for distribution under Your Name. These are designed to assist practitioners with vaccination plans.

PACKAGES

WESTERN TYPE	EASTERN TYPE
One treatment, 2-10 c.c. vials . . Code Westo	One treatment, 2-10 c.c. vials . . Code Easto
Five treatments, 10-10 c.c. vials . Code Wexal	Five treatments, 10-10 c.c. vials . Code Lewon

JENSEN-SALSBERY LABORATORIES, Inc.

KANSAS CITY, MISSOURI

PRINTING PRODUCTS CORPORATION—CHICAGO